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FACTORS IN THE RADIOLOGICAL DIFFERENTIAL DIAGNOSIS OF PYLORIC ULCER

1. THE PYLORIC ORIFICE SIMULATING AN ULCER

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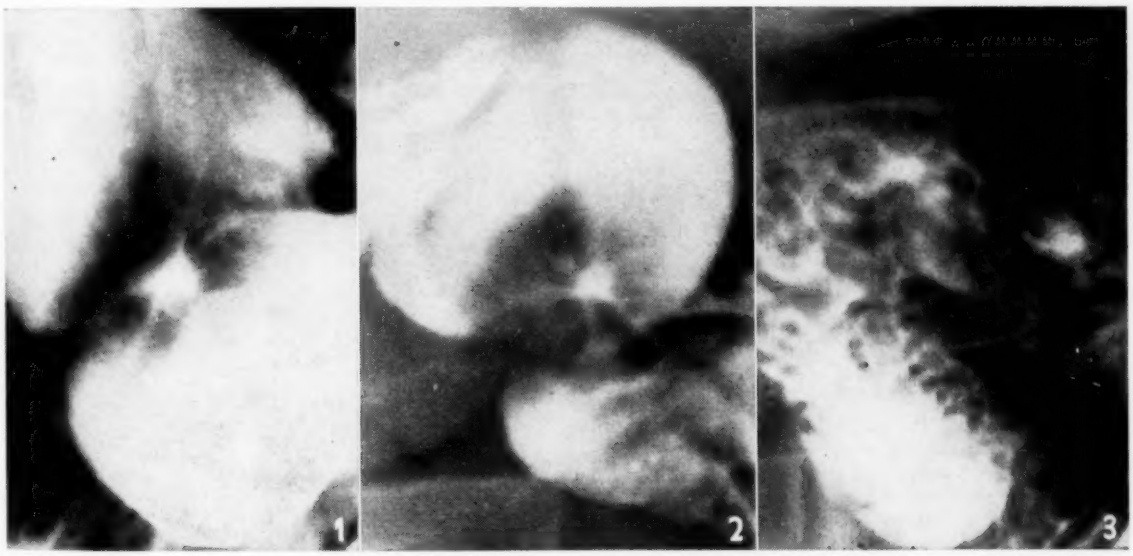
The radiological diagnosis of an active gastric ulcer depends on direct and indirect signs. The indirect signs *per se* are often inconclusive and, in practice, the diagnosis depends on the direct sign, viz., the demonstration of an ulcer cavity. This may present in two ways, as follows:

1. It may have the appearance of a barium-containing projection from the lumen. In this case the ulcer is seen in profile, i.e. sideways, on one of the margins of the organ.

2. It may be seen as a persistent dense fleck. Here the barium-containing ulcer cavity is seen *en face*, or end-on. The fleck can usually be demonstrated both in the mucosal views and in the filled stomach with graduated compression. By means of compression, the gastric walls are approximated and the barium in the lumen is pressed away, leaving only the barium in the crater to present as a dense fleck.

Depending on the situation of the ulcer it may, at times, be visualized in both ways. Generally speaking, an ulcer on one of the curvatures will present as a projection from the lumen. When situated on the anterior or posterior surfaces of the organ it is more likely to present as a constant fleck. If this is in the 'vertical' part of the stomach it is often possible to rotate the patient in such a way that the crater becomes visible on the 'sky-line', in which case it will also be seen in profile. Where the ulcer is located on the anterior or posterior walls of the 'horizontal' part of the organ such rotation is difficult, and the diagnosis has to depend on the demonstration of a constant fleck. A projection from the lumen may, of course, also be caused by a gastric diverticulum, but this can usually be differentiated from an ulcer by various characteristics such as its situation, rounded shape and narrow neck. Other causes of small projections from the lumen are protrusions between peristaltic waves (which may resemble an ulcer to a remarkable degree on single films) and collections of barium in protruding mucosal furrows.

As to the differential diagnosis of the fleck, it should be pointed out that an inconstant fleck may occur at a convergence of two mucosal furrows. Owing to the mucosal movements in the normal stomach, classically described by Forssell,[1,2] such a fleck will not be constant. In a series of exposures it should be possible to demonstrate these movements, with the consequent change in appearance or disappearance of the fleck. In cases of spasm of the prepyloric musculature, however, there is diminished movement of the muscularis propria. Owing



Figs. 1, 2 and 3. In each case there is a constant, dense fleck at the pyloroduodenal junction, surrounded by a radiolucent zone and radiating mucosal folds. A greater degree of compression was used in Fig. 3.

to the 'independent but coordinated movements'[1,2] of the muscularis propria and the mucosal membrane (muscularis mucosae), the movement of the latter will be proportionately diminished. In these cases the pseudo-niche may be more permanent and there may be difficulty in distinguishing it from a true niche. A constant fleck may, of course, also occur on the inner, ulcerated surface of a tumour. In these cases it is generally possible to demonstrate an additional irregular or rounded filling defect.

Apart from the constant fleck, other direct signs of gastric ulcer are a zone devoid of barium around the fleck, caused by mucosal swelling, and radiating mucosal folds, due to the puckering effect of the ulcer. This has been called the rosette appearance.[6]

The purpose of this short communication is to draw attention to 3 cases in which a constant fleck in the pyloric area, resembling an ulcer in various ways, was produced physiologically. It is an appearance that seems to be seldom mentioned in the differential diagnosis of pyloric ulcer. The majority of textbooks do not refer to it, although Schinz *et al.*[5] mention it, while Templeton[7] has an illustration of it, and calls it the 'pyloric star'. Meschan[4] notes that there may be a dimple of mucosa at the base of the bulb when the pylorus closes, in which barium may accumulate, giving rise to the appearance of a fleck.

CASE RECORDS

Case 1

The patient was a woman, aged about 38 years, seen at another hospital.

At the barium-meal examination there was a moderate spasticity of the prepyloric musculature. The pyloroduodenal axis was more horizontal than usual, so that films of this area could not be obtained in the left oblique position. The axis pointed to the right in the usual way, but its sagittal inclination was exaggerated. The result of this was that the caudal part of the prepyloric area was projected over the base of the bulb in the anteroposterior and right oblique positions. We did not succeed in separating these two overlying shadows. Compression of this area showed a constant dense fleck of barium at the pyloroduodenal junction, surrounded by a zone devoid of barium and showing radiating mucosal folds (Fig. 1). About 4 years before this examination a cholecystectomy had been performed.

At operation a fortnight after the barium meal, dense adhesions were found between the liver, pylorus and duodenum. It was thought that these were due to peptic ulcer, and a partial gastrectomy was performed. The resection specimen showed the changes of gastritis, but no active ulcer.

Case 2

At the barium-meal examination the findings were almost identical to those seen in case 1. It was reported that it was difficult to distinguish between an ulcer and a physiological condition in which the constant fleck was produced by the pyloric orifice, the round defect by the pyloric valve and the radiating folds by gastric and duodenal mucosal folds converging on the pylorus (Fig. 2). The films also showed a calcified hydatid in the liver. Ten years before admission a hydatid cyst had been removed from the liver.

At operation 3 days later, adhesions were found between the liver, stomach and duodenum. These were severed. An annular thickening was felt at the pylorus, and it was decided to do a partial gastrectomy. The gastric resection specimen showed an atrophic mucosa with disappearance of rugae in many areas. The histology was that of a chronic, atrophic gastritis. There was no sign of malignancy and no ulceration. The thickening felt at operation was not commented upon, but it was probably due to a degree of local pyloric hypertrophy.

Case 3

For some months we have been doing routine radiological examinations of the pyloroduodenal junction in cases where the stomach and duodenum had been proved normal at an earlier cholecystectomy. Case 3 was from that series and again showed a constant fleck, surrounded by a radiolucent zone and radiating mucosal folds at the pyloroduodenal junction (Fig. 3).

DISCUSSION

It is clear that in these 3 cases, in which the stomach and duodenum were proved free of ulceration and tumour, the constant dense fleck resembling an ulcer at the pyloroduodenal junction was produced by barium in the pyloric orifice. Because of the peculiar direction of the pyloroduodenal axis and moderate spasticity of the prepyloric area, the caudal end of the latter was projected over the base of the duodenal bulb and could not be separated from it. Consequently the pyloric orifice was seen end-on and presented as a constant fleck. In view of the spasticity of the prepyloric musculature, and probably also because of the adhesions between liver, stomach and duodenum in 2 of the cases, there was diminished rhythmic contraction and relaxation of the prepyloric area, and the fleck remained unchanged throughout the examination.

SUMMARY

Three cases are described in which a constant dense fleck of barium surrounded by a radiolucent zone and radiating mucosal folds, thus resembling an ulcer, was produced physiologically by the pyloric orifice. In these cases a peculiarity in the direction of the pyloroduodenal axis was present, and the prepyloric musculature was spastic. In 2 of the cases there were massive adhesions between the liver, stomach and duodenum.

2 A COMMON NORMAL PREPYLORIC MUCOSAL FURROW SIMULATING AN ULCER

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A small, ulcer-like projection on the lesser curvature of the prepyloric area of the stomach, is seen relatively often during barium-meal studies. The following case is an example:

Case 4

A man, aged 67 years, was admitted with obstructive jaundice of 1 month's duration. During the preliminary investigations the barium meal showed that the pyloric area of the stomach was relaxed throughout the examination, showing diminished rhythmic contraction. A small niche was seen, situated on the lesser curvature in this area just orally to the pyloric orifice (Fig. 4). It was visible at screening and was more or less constant on a number of films, and compression views showed what appeared to be a small, persistent fleck in this area. Owing to the inclination of the pyloroduodenal axis the area could not be adequately visualized in the left oblique position.

At operation 3 days later a papillary adenocarcinoma was

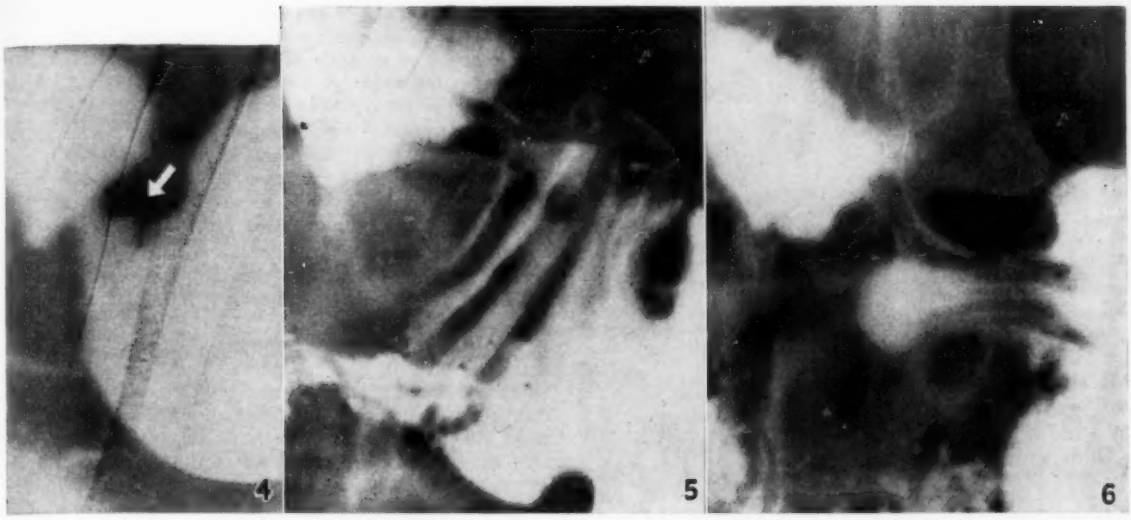

Fig. 4. Case 4. Small projecting niche on lesser curve orally to pyloric orifice.
Fig. 5. Case 5. Prepyloric area relaxed. Mucosal folds in lumen lie obliquely.
Fig. 6. Case 5. Prepyloric area contracted. Mucosal folds now lie longitudinally.

found at the ampulla of Vater, giving rise to obstruction of the common bile-duct. Although the pylorus felt slightly thickened, it was stated after careful inspection and palpation that the stomach was normal. A pancreaticoduodenectomy was performed.

In this case, then, it was shown that the ulcer-like projection was not due to an organic lesion in the stomach. In view of its relatively frequent occurrence, an attempt was made to determine the causal mechanism. Was it a fortuitous occurrence, or did it have an underlying physiological or anatomical basis? In order to find an answer to these questions, the prepyloric area was systematically examined in a number of normal stomachs.

It was seen that, normally, when the prepyloric area (canalis egestorius of Forssell[1,2] and Torgersen[8]) was relaxed or only partially contracted, the mucosal folds in the lumen run transversely or obliquely (Fig. 5, case 5). When the canalis contracts, the folds change in direction and come to lie longitudinally. With maximal contraction of the muscularis propria of the canalis, only longitudinal mucosal folds are seen in the lumen (Fig. 6, case 5).

Forssell and Torgersen showed that the movements of

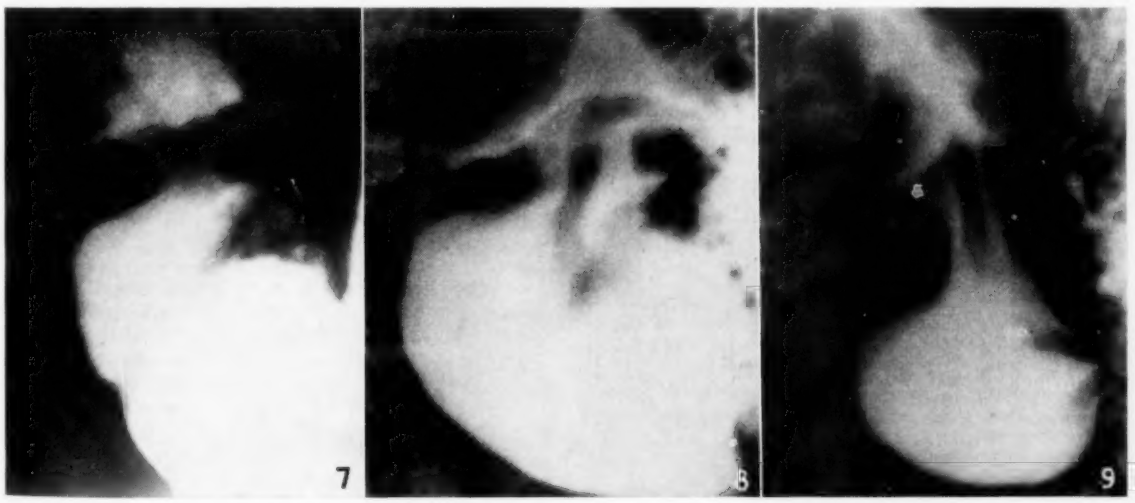

Fig. 7. Case 6. Prepyloric area relaxed. Prominent oblique fold with barium collection on its caudal side.
Fig. 8. Case 7. The barium projection resembles an ulcer niche. Prepyloric area relaxed.
Fig. 9. Case 7. Prepyloric area contracting. Folds change in direction and 'niche' disappears.

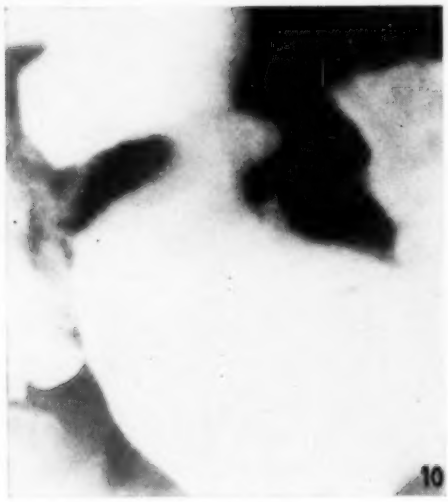

Fig. 10. Case 8. Ulcer niche projecting from lesser curve of pyloric orifice.

the intestinal muscularis propria and the mucosal membrane are independent but coordinated in the entire alimentary canal. One of the best examples of these independent but coordinated movements, then, is seen in the prepyloric area of the stomach where the folds run transversely or obliquely when this area is relaxed, and longitudinally when it is contracted.[3] It is a regular feature seen in all normal stomachs.[3]

Between the folds are barium-containing mucosal furrows. One of the oblique mucosal folds may be relatively prominent with relaxation of the canalis, causing a slight projection consisting of a collection of barium to lie in the furrow on its caudal side, between the fold and the pyloric orifice (Fig. 7, case 6). When the collection of barium in the furrow is a little more prominent, it may resemble an ulcer niche (Fig. 8, case 7). Note in Fig. 8 that the prepyloric area is relaxed and that an oblique fold can be seen orally to the pseudo-ulcer niche. When the muscularis propria contracts, the mucosal folds and furrows change in direction, come to lie longitudinally, and the 'niche' disappears (Fig. 9, case 7). In other words, the presence or absence of the mucosal niche is directly related to the stage of relaxation or contraction of the muscularis propria in this area.

That this 'niche' may resemble an ulcer niche in the pyloric orifice to a remarkable degree is seen in a case of pyloric ulcer (Fig. 10, case 8). In case 8 the niche was permanent, irrespective of the degree of contraction of the muscularis propria in the prepyloric area.

The films were all taken in the right oblique position. In the left oblique position the fold and furrow on the lesser curve are situated posteriorly and, generally speaking, are less prominent.

The fleeting character of the pseudo-niche, and the fact that it is present with relaxation of the canalis and disappears when the latter contracts, differentiates it from a true ulcer niche. The differentiation becomes difficult when the prepyloric area remains relaxed or in a state of partial contraction. In these cases the mucosal folds remain transversely or obliquely, and a pseudo-niche due to a mucosal furrow will tend to be permanent. This appears to be the state of affairs in case 4.

SUMMARY

An oblique prepyloric mucosal fold, with an accompanying mucosal furrow on its caudal side, is commonly seen in the normal stomach when the canalis egestorius is relaxed or partially contracted. The barium-containing mucosal furrow may present as a niche on the lesser-curve side. When the canalis contracts maximally the fold and furrow change in direction and come to lie longitudinally, and the 'niche' disappears. It has to be differentiated from pyloric ulcer.

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WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING SEPTEMBER 1960

Workshop for Comparative Neurology and Neuropathology, 1st Meeting, Berne, 28 - 29 November 1960. Prof. Dr. E. Frauchiger, Secretary, c/o Veterinary-Ambulatory Clinic, University of Berne, Neubruckstr. 10, Berne, Switzerland.

United Nations Educational, Scientific and Cultural Organization, Symposium on Radiobiology, Warsaw, November 1960. Polish Academy of Sciences, Section of Medical Sciences, Palac Kultury i Nauki, Warsaw, Poland.

Symposium on Pest Control by Radiation, India, 5 - 9 December 1960. International Atomic Energy Agency, 11 Kärtner Ring, Vienna 1, Austria.

Regional Symposium on the Use of Radioisotopes in the Study of Endemic and Tropical Diseases, Bangkok, 12 - 16 December 1960. International Atomic Energy Agency, 11 Kärtner Ring, Vienna 1, Austria. In cooperation with the World Health Organization.

European Council for Nuclear Research, 16th Session, Geneva, December 1960. Geneva 23, Switzerland.

Symposium on Detection and Use of Tritium in the Physical and Biological Sciences, Vienna, January 1961. International Atomic Energy Agency, 11 Kärtner Ring, Vienna 1, Austria.

International Federation of Resistance Movements, Medical Congress, Spring 1961. Castellezgasse 35, Vienna II, Austria.

International Congress on Analytical Chemistry, Budapest, 16 - 21 May 1961. Magyar Kémikusok Egyesülete, Szabadságtér 17, Budapest V, Hungary.

International Meeting on High Powered Radiotherapy. (Convegno Internazionale sulla Radioterapia con Elevate Energie), Turin, 10 - 11 June 1961. Minerva Medica, Corso Bramante 83 - 85, Turin, Italy. Part of the 4th International Medical-Surgical Meetings, 3 - 15 June.

World Congress on Odontotechny, Turin, June 1961. Minerva Medica, Corso Bramante 83 - 85, Turin, Italy. Part of the 4th International Medical-Surgical Meetings, 3 - 15 June 1961.

VAN DIE REDAKSIE : EDITORIAL

DIE DRANG NA GEWELD

'n Verskynsel in die hedendaagse samelewing wat elke geneesheer in die loop van sy alledaagse werk moét opval, is die vernietigende, alomteenwoordige drang na geweld. Daar is haas nie 'n gebied van die lewe waar die sadistiese smaak vir geweld nie ingesypel het en reeds deur 'n ontstellende groot persentasie mense min of meer as die normale gang van sake aanvaar word nie.

Die geneesheer kom die uitings van geweld teë by die slagoffers van gewelddadige aanrandings, by pasiënte wat ná onbeteuelde gevegte na die noodgevalle-afdeling van 'n hospitaal gebring word, by mishandelde kinders wie se ouers voor die hof gedaag word weens hul wrede optrede teenoor die kinders. Ook op die gebied van menslike verhoudinge kom die geneesheer dikwels geweld in 'n meer verborge, subtieler vorm teë. Liefdeloosheid neem so maklik die sadistiese vorm van emosionele geweld aan.

Die drang na geweld lê diep in die mens. En elke mens dra die stempel van die menslike aard. Daar is oomblikke in elkeen van ons se lewe, sê Schopenhauer, wanneer die boosheid van ons natuur ons tot moordenaars kon maak. Dit is 'n drang waarteen elke mens in sy strewe na selfbeheersing en geestelike groei gedurig moet waak. Die tragiek lê daarin dat die stryd nie vir almal gelyk is nie. Een mens ondervind meer dwarsboming as 'n ander. Dwarsboming kan so maklik tot 'n gevoel van verontregting lei en uit verontregting word wraaksug gebore. En die bevrediging wat geweld bied, voed die wraaksug.

Die smaak vir geweld word ongetwyfeld deur 'n gevoel van verveling gevoed. In hierdie gemeganiseerde eeu ken baie min mense die vreugde van skeppende arbeid. Die gevoel dat 'n mens in die persoonlike sin 'n plek het in die beskaafde staat word flouer en flouer; en waar daardie sin van persoonlike verantwoordelikheid in duie stort, word die mens 'n ontwortelde sonder tuiste en sonder anker. Só word hy by uitstek vatbaar vir die verdowing van geweld. Vir die geneesheer wat gedurig met mense te doen het, is dit van belang om op sommige van dié faktore te let wat vandag tot die kultus van geweld in die samelewing bydra.

Groot kweekplek vir geweld in die hedendaagse samelewing is sonder twyfel die bioskoop. Daar word selde 'n rolprent vertoon waarin geweld nie 'n rol speel nie. 'n Rolprent waarin daar nie 'n verbete vuisgeveg voorkom nie, is 'n hoë uitsondering. Moorde word gepleeg, brand word gestig, daar word sonder inhibisie kruis en dwars geskiet en mense sterf op aaklige wyse sonder dat iemand ooit protesteer. Geweld word nie as pornografie bestempel nie en ouers gee skynbaar nie om dat hul kinders Saterdagmiddag na Saterdagmiddag op geweld getrakteer word nie. Die bioskope loop nooit leeg nie en niemand spreek op grond van die geweld kritiek op die rolprente uit nie.

'n Ander vrugbare medium vir die kweek van geweld is die strookprent wat vandag vir baie mense die enigste leesstof is waarvoor hulle kans sien. Verreweg die belangrikste inhoud van die strookprente, veral van die gekleurde strookprente wat in die naweek-koerante verskyn en die strookprente wat die kinders verslind, is niks anders as growwe, onverbloemde geweld nie. Die smaak vir dié soort leesstof bestaan en dit word naarstigtlik gevoed.

In die intellektuele kringe is daar 'n ander teken van die tyd: 'n fyn, gekultiveerde geesdrif vir stiergevegte. Dit word vandag as 'n teken van hoë geestesontwikkeling beskou om 'n smaak vir die stiergeveg te bely. Daar is baie mense wat dit as 'n hoogtepunt van hul lewe beskou om 'n pelgrimstog na Spanje te maak net om 'n stiergeveg by te woon. En dié wat nie die stiergeveg kan bywoon nie, lees diepsinnige boeke oor die mistieke kultus van die stiergeveg. Maar in die werklikheid is dit niks anders as 'n verdere vorm van verslawing aan geweld nie. Die Romeine het eers gewoond geraak om die openbare slagting van diere te aanskou en dit as 'n vorm van vermaak te waardeer. Daarna was dit makliker om die slagting van mense te aanskou en dít as 'n vorm van vermaak te waardeer. Die dieptes van ontaarding word nie in 'n enkele stap bereik nie.

Dit is opvallend dat die misdadiger as held vandag 'n heel besondere plek het in die hedendaagse literatuur. Meer as een tronkvoël het al beroemdheid verwerf met sy lewensverhaal. Artikels en verhale oor misdaad het altyd 'n gretige leserskring. Sodra daar 'n groot hofsaak in 'n koerant gerapporteer word, styg die sirkulasie van die koerant. Daar word nie 'n moordenaar verhoor nie of daar is 'n groot skare vroue wat die hofsitting kom bywoon. Dit is asof die gedwarsboomde, verveelde mens bevrediging soek in sy bure se toiingrigheid, swakheid en onvolkomenheid. In vroeër eeue was daar die ketterjagte, die uitruik van hekse, die aanskoulike dood op die brandstapel. Vandag drom die skares saam by die hofsitting waar 'n moordenaar ter dood veroordeel word. Die donker drang is nog daar. Ons het nog nie so beskaafd geword dat ons die smaak vir geweld verloor het nie.

Die beheptheid met geweld kom ook tot uiting by die groot gehore wat boksgevegte bywoon. Mense word ernstig beseer en in sommige gevalle selfs gedood in die bokskryt, maar sodra daar sprake is dat boks as ‚sport' afgeskaf moet word, kom die aanhangers daarvan in heftige opstand. As 'n mens die primitiewe bloedlus van die mens in sy naakte vorm wil aanskou, moet jy net die gehoor by 'n boksgeveg gaan dophou.

Vir die geneesheer wat hom beywer vir die liggaamlike en geestelike welsyn van sy pasiënte, is dié probleem van geweld 'n vraagstuk van wesentlike belang. In sy benadering daarvan sal hy steeds sterker tot die insig kom dat die mens se grootste behoefte 'n behoefte aan betekenisvolle inhoud in sy lewe is. Die geneesheer wat sy pasiënt deur simpatieke begrip en wysheid kan help om daardie sinvolle inhoud vir sy lewe te vind, hetsy deur meer positiewe aandag aan persoonlike verhoudinge, hetsy deur 'n verandering van werkkring, 'n verbreding van belangstelling, 'n stokperdjie waaraan hy sy geesdrif kan wy, 'n manier waarop hy die gemeenskap kan dien, of 'n geleentheid om nuwe vriendskappe te sluit — die geneesheer wat dít kan doen, sal in der waarheid kan voel dat hy 'n oorwinning behaal het oor die vernietigendste mag in die wêreld — die mag van geweld.

SUDDEN DEATH IN EARLY LIFE

The subject of sudden and unexpected death in infancy has been studied by a number of authors.[1] It is dealt with in a paper recently read by Morrison[1] before the Pediatric Society of North-Eastern Pennsylvania and published in the *Journal of the American Medical Association*. He records that, in the six and a half years ended 30 June 1959, of the one hundred and eighty-nine paediatric deaths at the Geisinger Memorial Hospital, seventeen were classified as unexpected, according to the following definition of the term: 'The death of a child who was thought to be in good health or whose terminal illness appeared to be so mild that the possibility of a fatal outcome was not anticipated'.[2] All the seventeen children were either moribund when cause for alarm was recognized (fourteen) or were found dead (three). In none of them had symptoms been present for more than forty-eight hours. Their ages ranged from one day to thirty months; ten were between two and eight months old, five were aged one month or less (including one who was one day old), and the other two were twenty months and thirty months old.

A study was made of the clinical, laboratory and necropsy findings in the seventeen cases. The following procedures were carried out in all of them within ten minutes of the pronouncement of death: Cultures from heart blood; nose, throat and anal cultures; lumbar puncture, followed within thirty minutes by cultures from the centrifuged CSF sediment, and examination of the CSF for cell, protein, and sugar content. A complete autopsy was performed in each case, including gross and microscopical examination. The seventeen cases were classified according to the diagnoses that were made from these studies.

It was concluded that seven of them died of infective conditions, viz. meningococcal septicaemia (three cases), pneumococcal meningitis, meningitis due to *Haemophilus influenzae*, staphylococcal septicaemia, and croup resulting from laryngotracheitis and oedema of the glottis due to *Haemophilus influenzae*, type B.* Under treatment with antibiotics and sulphonamides these conditions are ordinarily considered to be curable. All seven showed signs of illness from two to forty-eight hours before death, and Morrison expresses the opinion that several of these children could possibly have been saved.

In three cases a postmortem diagnosis of endocardial fibro-elastosis was made. The cause of this condition, which is said to be always fatal, is not known; it is commonly associated with other forms of congenital heart disease. The three cases were less than a month old, and (like the rest of the series) they all died within forty-eight hours of the onset of symptoms. If they could have been diagnosed clinically the fatal conclusion would not have been prevented. This applies also to another patient in the series, who died at the age of twenty-nine hours and was found at autopsy to be a case of congenital heart disease with absence of coronary ostia and tricuspid and aortic valve and hypoplasia of the right ventricle.

In three other cases pulmonary oedema was the only abnormal finding after death. One of them was found dead in his cot after minor and non-specific symptoms of forty-eight hours' duration and the other two had been in apparently normal health when they were unexpectedly found dead in the cot. In a fourth case found dead in apparently normal health (aged eight months) pulmonary oedema was the only anatomical finding, but a coagulase-negative staphylococcus was cultured from the blood and electrophoresis showed only 0·081 g. per 100 ml. of gamma globulin in the blood, as compared with the observed normal range of 0·54 - 1·03 g. per 100 ml. at the age of seven to eleven months. (It has been suggested that a lowered antibody level or hypogammaglobulinaemia may be a factor in unexpected death during infancy, leading to rapid death from an ordinary minor infection.)

The significance of pulmonary oedema in infants who die suddenly remains undetermined. In Morrison's series it was also found in some of the infants in whom the cause of death was in fact identified.

The series of seventeen cases is completed by a boy (the twenty-months-old case) known to be suffering from nephrosis for three months before he was admitted to hospital for a (second) course of treatment with ACTH, where he died suddenly, possibly, Morrison suggests, as the result of this treatment, and a baby aged two months whose death resulted from birth injury and intramedullary haemorrhage.

It is noteworthy that not one of the cases of sudden death was attributed to status thymicolymphaticus. Excluding one six-month-old infant who died with meningococcal septicaemia and bilateral adrenal haemorrhage and had a thymus gland weighing 60 g., the weight of the thymus glands in the series ranged from 4·4 to 38·0 g. (as compared with Boyd's figures of 4·3 to 31·0 g. for the normal thymus gland in the first year of life.[3])

Nor were any of the cases attributed to mechanical suffocation. In the literature Morrison[1] finds little satisfactory evidence of deaths from suffocation with bedclothes and pillows. Davison[4] reported on three hundred and eighteen cases in which, on the death certificates, infant deaths had been attributed to suffocation. He was only able to corroborate this diagnosis in twenty-five cases, and in eighteen of these the baby was sleeping in bed with another person; the death might therefore have been caused by overlying.

Two main conclusions are to be drawn from Morrison's study of sudden and unexpected deaths in early life: (1) A proportion (in his series forty-one per cent) of the cases are due to infection and with due care might be diagnosed during life and possibly saved. The meningococcal infection is a well-known cause of sudden death, not only in young children, and it should especially be borne in mind when cerebrospinal fever is prevalent. (2) The problems presented by the cases characterized by endocardial fibro-elastosis, and those in which pulmonary oedema is the only finding, remain unsolved and are subjects for further investigation.

* This case of croup occurred in the child (a boy) aged thirty months. The type B strain differs from the *H. influenzae*, ordinarily found in the nose and throat of healthy persons, in possessing a capsule.

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SOME OBSERVATIONS ON THE ATRIAL SOUND*

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The atrial sound, also known as the fourth heart sound or presystolic gallop, has been defined[2] as an audible vibration occurring more than 0·07 seconds after the beginning of the P wave of the simultaneous electrocardiogram (ECG) but preceding the onset of the QRS complex. It has been shown[7] that, unrelated to any alteration in the P-R interval, the position of the atrial sound varies in relation to the first heart sound and to the simultaneous ECG.

It is the purpose of this paper to demonstrate this variation in the timing of the atrial sound and to discuss some of the clinical implications resulting from this variability.

METHODS AND OBSERVATIONS

Low-frequency phonocardiographic (PCG) tracings on an apparatus similar to that recommended by Leatham[9] were used throughout. Standard lead II was used for the simultaneous ECGs. In order to determine the position of the atrial sound, the distance from the beginning of the P wave to the commencement of the atrial sound was used. This measurement was suggested by Duchosal[3] and is referred to as the P-G interval (G=gallop).

Details of the variation in timing of the atrial sound are described more fully elsewhere.[7] For the purpose of this paper they are briefly referred to here as follows:

1. *Effect of clinical improvement and deterioration.* Serial PCGs on patients with ischaemic or hypertensive heart disease have shown that with clinical improvement the P-G interval increases so that the atrial sound approaches the first heart sound and may eventually fuse with it (Fig. 1). Likewise, clinical deterioration results in a decrease of the P-G interval, the atrial sound occurring earlier in the cardiac cycle and consequently further away from the first heart sound (Fig. 10). These changes may occur in a few minutes or may take many weeks or months, depending on the time and extent of the clinical improvement or deterioration. For example, the atrial sound took about 4 weeks to merge with the first heart sound in a man recovering from a severe myocardial infarction (Fig. 1), whereas in another patient an attack of ischaemic pain at rest caused the atrial sound to move in a few minutes from a position in which it was fused with the major components of the first sound to one considerably earlier in the cardiac cycle (Fig. 6). A similar movement of the atrial sound has been shown to occur in severe hypertensive patients when the blood pressure is rapidly reduced by the administration of intravenous hexamethonium. As the blood pressure is allowed to rise again the P-G interval shortens and the atrial sound returns to its original position (Fig. 2).

2. *Effect of rest and emotion.* Some movement of the atrial sound towards the first heart sound, usually not more than 0·02 or 0·03 seconds, has frequently been noted in patients with hypertensive or ischaemic heart disease after rest lasting from a few minutes to an hour (Fig. 3). Emotional factors probably play a large part in this; the P-G interval may quickly shorten again on the introduction of an intravenous needle or similar procedure.

3. *Effect of respiration.* Whereas respiration produces no effect on the P-G interval in patients with hypertension or ischaemia, the atrial sound of cor pulmonale both increases in intensity and occurs earlier in the cardiac cycle during inspiration as compared with expiration (Fig. 4).

4. *Effect of various procedures.* It has already been mentioned that the intravenous administration of hypotensive drugs will increase the P-G interval in hypertensive patients. The inhalation of amyl nitrite has a similar effect in hypertension (Fig. 5), though a change in the P-G interval is seldom seen in cases with ischaemic heart disease following inhalation of this drug. Venous cuffing will prolong the P-G interval in both ischaemia and hypertension, the atrial sound returning to its previous position a few seconds after the cuffs are released.[7] Carotid-sinus pressure will often diminish the intensity of an atrial sound.[14] If the P-R interval is prolonged by this manoeuvre the atrial sound is obviously more widely separated from the first heart sound. The P-G interval, however, remains virtually unchanged.

5. *Effect of movement of atrial sound on the first heart sound.* The major components of the first sound, which are probably due to mitral and tricuspid valve closure,[10, 15] become louder as the P-G interval lengthens. This is best illustrated when the atrial sound approaches the first heart sound with rest (Fig. 3), for other factors, such as the specific effect of amyl nitrite, which invariably increases the intensity of the first sound,[1] or the possible increased intensity of the first sound due to a tachycardia associated with the hypotensive effect of hexamethonium, are then excluded. The change in P-G interval due to an alteration of the P-wave shape, but with the same heart rate and P-R interval, confirms this effect on the intensity of the major components of the first sound (Fig. 7).

DISCUSSION

In severe cases of ischaemic or hypertensive heart disease the P-G interval may be as short as 0·08 seconds. With improvement in the cardiac state, or lowering of the blood pressure in hypertensive patients, the P-G interval may increase to 0·20 seconds or more — depending to an extent on the length of the P-R interval. At this stage the atrial sound may have fused with the first heart sound and will then occur after the Q of the simultaneous ECG (Fig. 1). It is now indistinguishable, both clinically and by PCG, from a 'split' first sound in normal subjects in whom the earlier component is low pitched (Fig. 8). When an atrial sound has moved to this position, in which it occurs after the Q, it is, by definition, no longer an atrial sound and is better referred to as an atrial component of the first heart sound.[6]

The movement of the atrial sound can usually be appreciated on clinical auscultation. When the P-G interval is short and the atrial sound consequently widely separated from the first heart sound, the cadence is one of gallop rhythm, whereas, with improvement and thus lengthening

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of the P-G interval, the atrial sound approximates to the first sound and the cadence resembles that of splitting of the first heart sound.

It will be apparent, however, that the length of the P-R interval will also affect the position of the atrial sound in relation to the first heart sound and consequently may alter the cadence on clinical auscultation. In one study of the heart sounds in hypertension,[3] an atrial sound was present in at least 50% of cases and the average P-G interval for the series was 0·145 seconds. It is obvious that in any one patient in that group with a P-R interval of, say, 0·20 seconds, the atrial sound would occur earlier in relation to the first heart sound than in another patient with the same P-G interval but a P-R interval of, say, 0·15 seconds. In the first patient the cadence would be that of gallop rhythm, whereas in the second the atrial sound would be closer to the first heart sound and the cadence would be more like that of a 'split' first sound.

Weitzman[16] observed that the atrial sound, as shown on a PCG, often consists of an early 'inaudible' component and a second main, or 'audible', component. The inaudible component occurs 0·08 - 0·14 seconds after the beginning of the P wave[16, 7] and is unaffected by any change in clinical state or by the procedures mentioned above which alter the P-G interval. It may also be recorded on a PCG in normal patients. Weitzman[16] considered that this inaudible component was caused by the actual muscular contraction of the atrium. It certainly cannot be related to ventricular filling, which is probably the cause of the main audible component, since it can be recorded with atrial contraction during ventricular systole in patients with complete heart block (Fig. 9), when ventricular filling would clearly be impossible. It is to the second, and audible, component of the atrial sound to which all the above observations refer. This sound is never heard or recorded in a normal heart with a normal P-R interval. In, however, a heart otherwise normal but with a prolonged P-R interval, an atrial sound may be clinically audible and the P-G interval will then be anything from 0·21 to 0·28 seconds.[7] A similar long P-G interval is also seen when atrial contraction occurs in early diastole in patients with complete heart block. An atrial sound due to first-degree heart block in an otherwise normal heart is not moved by the inhalation of amyl nitrite or the application of venous tourniquets.[7]

An approximate estimation of the P-G interval can be made on clinical auscultation just as an experienced observer can assess whether the opening snap of mitral stenosis is occurring early or late. However, it will be apparent from the above that any clinical assessment of the P-G interval is liable to be hopelessly inaccurate unless the P-R interval is known. Clinical assessment of the P-G interval must depend on estimating, by the cadence of the sounds, the distance between the atrial sound and the first heart sound. This interval, however, could theoretically be the same in a patient with a normal heart except for first-degree heart block, a patient with a P-R interval of about 0·20 seconds with a moderate P-G interval, and a patient with a P-R interval of 0·14 seconds but with severe cardiac decompensation and consequently a short P-G interval (Fig. 11). An approximate clinical estimation of the P-R interval can sometimes be made, provided no valvular disease is present, by judging the intensity of the first heart sound. But it has already been mentioned that the position of the atrial sound in relation to the first heart sound has an effect on the intensity of the latter (Figs. 3 and 7) and thus this method must inevitably be very inaccurate. A recent ECG, however, is usually available; so when the P-R interval is known, some assessment of the P-G interval can be made on clinical auscultation. A subsequent change in P-G interval, following improvement or deterioration in cardiac function, can then usually be elicited without difficulty.

The decrease in the P-G interval during inspiration which is seen in patients with atrial sounds originating from the right side of the heart (Fig. 4), suggests that the increased venous return during this phase of respiration places an added strain on these chambers. A right atrial sound is best heard over the lower sternal area or in the epigastrium and will obviously be more easily audible during deep inspiration. In this phase of respiration it is both louder and more widely separated from the first heart sound. On some occasions a right atrial sound is audible only during deep inspiration, while with normal respiration it might be confused with a 'split' first sound or be completely inaudible. The movement of a right atrial sound with respiration, the P-G interval of a left atrial sound remaining unchanged, may sometimes be a help in determining from which ventricle a gallop is originating.

The exact mechanism of the atrial sound is still uncertain. It often occurs too late in relation to the P wave to result from the actual muscular contraction of the atrium and, in any event, the sound is too loud for this to be likely. However, the muscular contraction could well be the cause of the early 'inaudible' component which is sometimes recorded on a PCG.[16] Potain[13] postulated that the atrial sound resulted from sudden tension of the ventricular wall following the inflow of blood in atrial systole. Kuo *et al.*,[8] by recording simultaneous atrial and ventricular pressures, showed that the atrial pressure is higher than the ventricular at the time that the atrial sound occurs. It seems improbable, therefore, that the atrial sound can be attributed to closure of the atrioventricular valves — the theory originally suggested by Lewis[11] and still favoured by some.[4]

It seems likely that the atrial sound is associated with ventricular filling resulting from atrial systole, but that resistance to this ventricular filling is increased. The actual sound heard could well, as suggested by Potain,[13] be caused by a sudden rise in tension of the ventricular wall. In patients in heart failure, the early atrial sound could then be due to a raised end-diastolic pressure favouring a rapid rise, after the atrial contraction, to the tension necessary to produce the sound. Clinical improvement, the application of venous tourniquets, or the lowering of the blood pressure, could then be expected to delay the atrial sound by lowering the end-diastolic pressure in the ventricle.

The different effects of amyl nitrite on the P-G intervals in hypertensive and ischaemic heart disease can possibly also be explained on this mechanism. Amyl nitirite causes a temporary decrease in peripheral resistance and hence a fall in blood pressure. This may be sufficient reason to account for the prolongation of the P-G interval in hyper-

tensive patients (Fig. 5). In ischaemic heart disease, however, the P-G interval is usually unchanged.[7] Possibly in this condition the temporary drop in peripheral resistance does not result in the ventricular end-diastolic pressure falling, and hence prolongation of the P-G interval, because the ventricular muscle is itself inadequate and the decreased peripheral resistance makes little or no difference to the inefficient contraction of the ventricle.

The significance of an atrial sound in any particular patient can be assessed only in relation to the nature of the underlying heart disease and the length of the P-G interval. An atrial sound in a patient with first-degree heart block and a P-G of not less than 0·21 seconds is of little significance. In such a case, of course, the cause of the heart block must, if possible, be ascertained and the prognosis assessed accordingly. In a series of hypertensive patients it was noted that only when the P-G interval was short (0·08 - 0·12 seconds) were moderate or severe cardiac symptoms present.[2] Hypertensive patients with mild cardiac symptoms or none, usually had a P-G interval of 0·15 - 0·20 seconds. Miles,[12] Weitzman[16] and Evans[5] have all agreed that atrial sounds occur in asymptomatic hypertensive patients, but it seems probable that the P-G interval is seldom less than 0·15 seconds in this group. Thus, if the blood pressure can be maintained at a level at which the P-G interval is long, then the prognosis as far as the heart is concerned is probably good. The presence of an atrial sound in normotensive patients with ischaemic heart disease is probably of more serious significance. In this condition it seems likely that the atrial sound implies inadequate ventricular function, and that, even with a relatively long P-G interval (0·15 - 0·20 seconds), symptoms of cardiac decompensation are likely to exist.

SUMMARY

The atrial sound is an audible vibration dependent on atrial contraction, which on a phonocardiogram (PCG) occurs more than 0·07 seconds after the beginning of the P wave of the simultaneous electrocardiogram (ECG) but precedes the onset of the QRS complex.

The position of the atrial sound is shown to vary in relation to the first heart sound and to the simultaneous ECG. This movement of the atrial sound, which is unrelated to any alteration in the P-R interval, has been studied in patients with hypertension, ischaemic heart disease, and cor pulmonale.

With improvement in cardiac function, the atrial sound approaches the first heart sound and may eventually fuse with it. When an atrial sound has moved towards the major components of the first sound to the extent in which it occurs after the Q wave of the simultaneous ECG, it is referred to as an atrial component of the first heart sound. In this position, it is clinically and by PCG indistinguishable from a 'split' first sound in normal patients in whom the first component of the 'split' is low pitched.

Some factors which cause movement of the atrial sound are described and discussed. These include clinical improvement and deterioration, rest and emotion, hypotensive drugs, the application of venous tourniquets, amyl nitrite inhalation, and respiration in patients with right atrial sounds in cor pulmonale.

The exact mechanism of the atrial sound is still uncertain. There is often an early 'inaudible' component, recorded on a PCG, which is possibly due to the actual muscular contraction of the atrium. The main 'audible' component probably results from increased resistance to ventricular filling at the time of atrial systole.

The significance of an atrial sound depends on both its position and the nature of the underlying heart disease. A clinical assessment of the position of the atrial sound can be made provided the P-R interval is known. It is thought that the presence of an atrial sound in ischaemic heart disease is of more serious significance than an atrial sound in a similar position in hypertensive heart disease.

I am grateful to the Editor of the *British Heart Journal* for permission to publish Figs. 1-10 inclusive.

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FIGURES 1 - 11

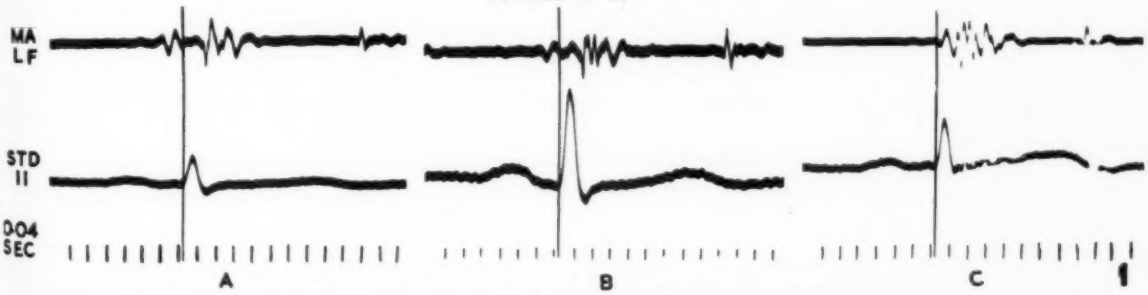

Fig. 1. Prolongation of P-G interval with improvement after myocardial infarction.
A. Phonocardiogram performed within 1 week of myocardial infarction. P-R interval 0·17 sec. P-G interval 0·10 sec.
B. Phonocardiogram about 2 weeks later. P-R interval 0·17 sec. P-G interval 0·13 sec.
C. Phonocardiogram about 5 weeks after myocardial infarction. P-R interval 0·17 sec. P-G interval 0·17 sec.
Atrial sound no longer precedes Q wave of ECG and is now an 'atrial component of first heart sound' — see text. In this position it is very similar to a normal heart — compare Fig. 9.
In this and other figures, the line drawn through the Q wave serves to illustrate the movement of the atrial sound.

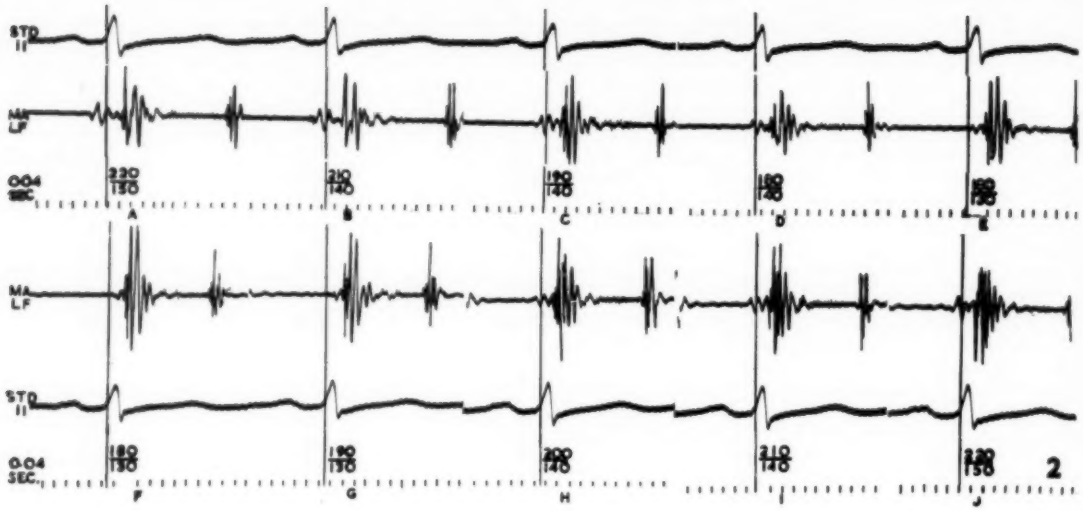

Fig. 2. Prolongation of P-G interval following intravenous hexamethonium in hypertensive patient. P-G shortens again when blood pressure is allowed to rise. Note that in tracings E, F and G, the atrial sound has become an atrial component of the first heart sound.

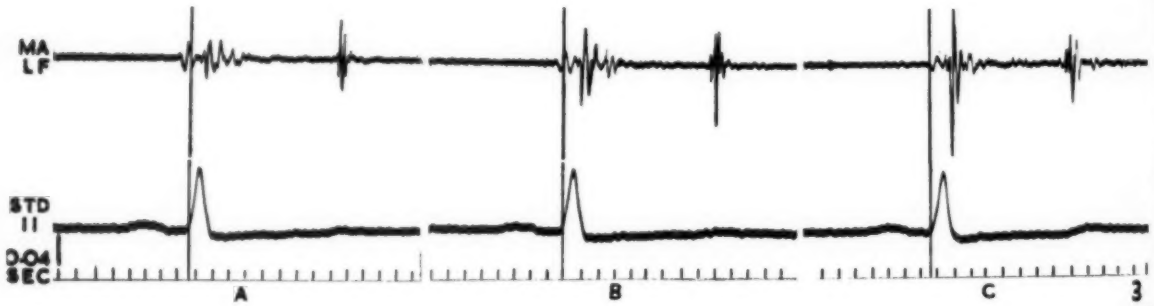

Fig. 3. Prolongation of P-G interval during rest for a period of about 1 hour. The increased intensity of the first heart sound as the P-G lengthens is clearly shown. The P-R interval is unchanged throughout.

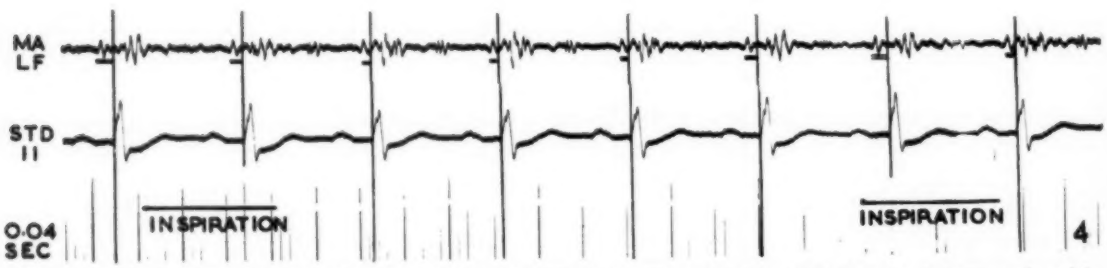

Fig. 4. Movement and change in intensity of the atrial sound in cor pulmonale. P-G interval shorter, and atrial sound louder, during inspiration, compared with expiration. The position of the atrial sound is readily seen by comparing its relationship to the line drawn through the Q of the ECG.

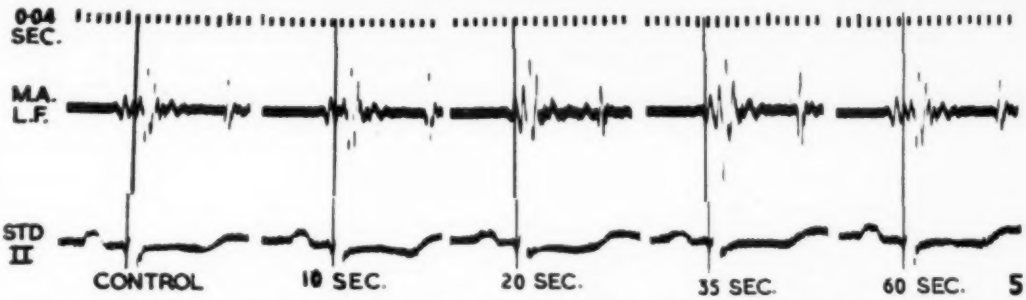

Fig. 5. The alteration of P-G interval in a hypertensive patient after the inhalation of amyl nitrite. Note also the increased intensity of the first heart sound in tracings taken 20 and 35 seconds after inhalation of the drug.

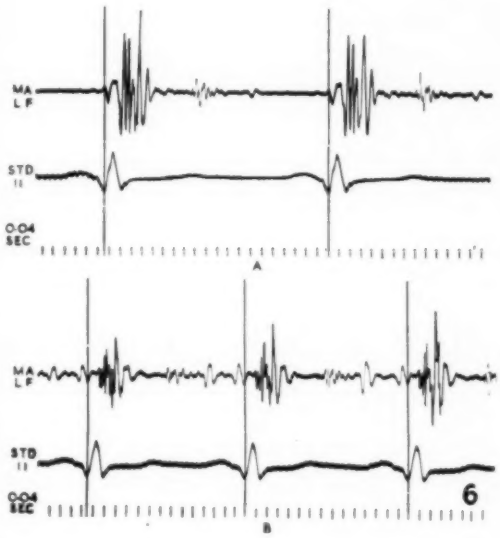

Fig. 6. Patient with ischaemic heart disease; showing shortening of P-G interval during an attack of angina at rest. (A) Before pain. (B) During pain. In both tracings a third heart sound is also present.

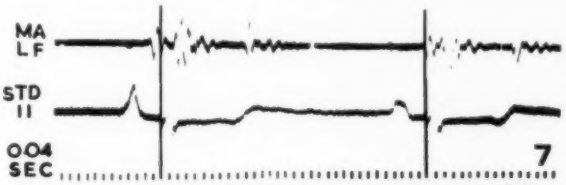

Fig. 7. Alteration in P-wave shape, but with same P-R interval. The first heart sound is louder in the complex on the right, where the atrial sound is delayed (i.e. prolonged P-G interval).

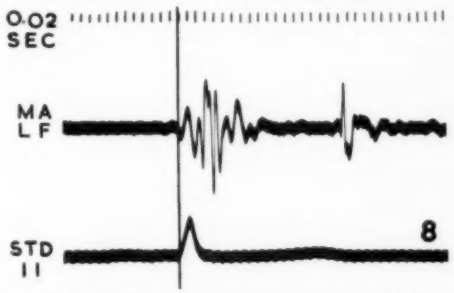

Fig. 8. Phonocardiogram of normal patient aged 30 years, with 'split' first heart sound in which the earlier component is low pitched. The beginning of this low pitched component is seen to be coincidental with the Q of the ECG. (Compare with Fig. 1 C). The P wave is poorly demonstrated but P-R interval was about 0·18 sec.

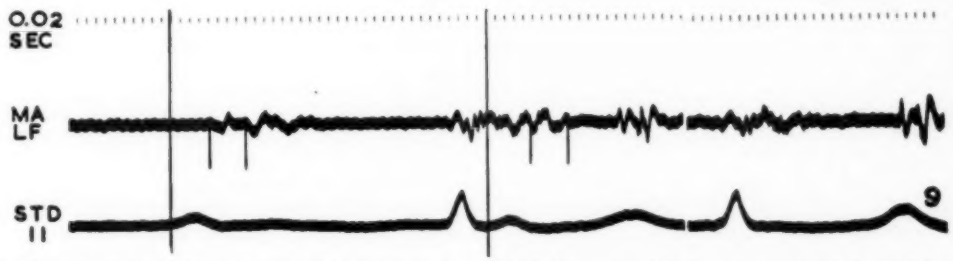

Fig. 9. Phonocardiogram in a patient with complete heart block demonstrating 2 components of the atrial sound. A long vertical line is drawn through the beginning of the P wave and the short vertical lines show the commencement of the two components of the atrial sound. It can be seen that the second component disappears when atrial contraction occurs during ventricular systole. The tracing on the right shows a flat base line between the first and second heart sounds when no P wave is present.

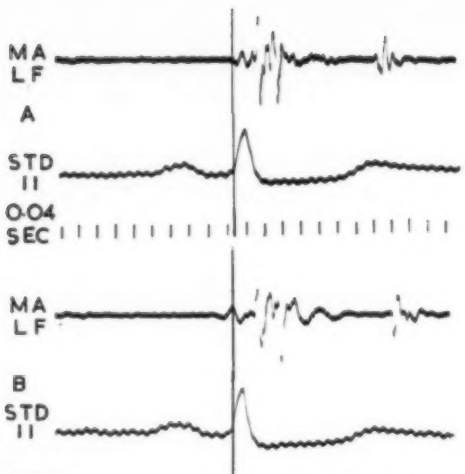

Fig. 10. Shortening of P-G interval over a period of several months in a hypertensive patient who refused therapy. (A) P-R=0·17 sec.; P-G=0·17 sec. (B) P-R=0·17 sec.; P-G=0·15 sec. (C) P-R=0·17 sec.; P-G=0·11 sec.

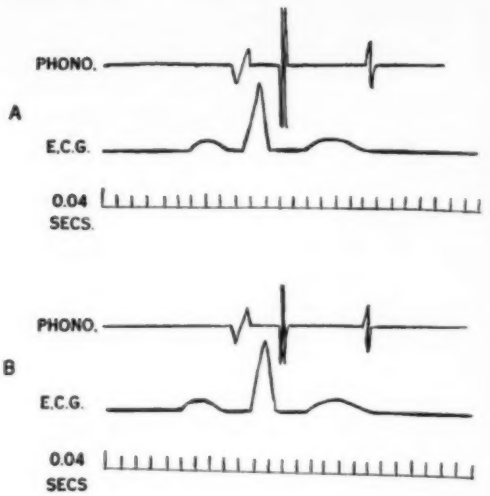

Fig. 11. Diagram to illustrate the necessity of knowing the P-R interval before the P-G interval can be clinically assessed. In the three examples shown, the atrial sound always precedes the first heart sound by 0·14 seconds. (A) P-R 0·14 sec.; P-G 0·10 sec. (B) P-R 0·20 sec.; P-G 0·16 sec. (C) P-R 0·30 sec.; P-G 0·26 sec. The intensity of the first heart sound has been decreased in (B) and (C) on account of the prolongation of the P-R interval.

A REVIEW OF A PHYSICIAN'S WORK IN A COMMUNITY MEDICAL SERVICE*

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I present this article as physician to an industrialist group which provides a complete medical and dental service for its employees and their families. This service, which now includes 15 full-time general practitioners and 12 full-time or part-time specialists, is responsible for the European working community of a steel works and its related industries, and their families. The people cared for numbered 8,000 in 1950, when they constituted 80% of the total population of the town in which they live, and 20,000 in 1958. Today they constitute 65% of the total population. The remainder of the European population of the town, not directly associated with the steel industry, and all the non-Europeans, are attended by the private practitioners of the town.

The material on which the article is based is a review of the office consultations of the physicians of this service in 3,132 cases.

The information that is offered was gathered not with a view to laying down any specific principles, but simply to extend the figures available in this country for that most difficult and valuable study in medical practice, the natural occurrence and pattern of disease. It is probably only by group reviews such as this that the general pattern of disease will be defined, the importance of various forms of research assessed, the natural history and prognosis of individual pathologies followed up, and their modification by our current methods of treatment estimated.

The review is subject to certain local difficulties and limitations, which must be defined. For instance, in valuing this study against South Africa as a whole one must remember that the population under survey is a comparatively young one and entirely European. The industry is a young one,

* Paper presented at the Congress of the Association of Physicians of South Africa (M.A.S.A.), Cape Town, January 1959.

and the workers in a young industry are young themselves, the 50 - 60 group are not numerous, and pensioners are few. Our figures will not therefore reflect the situation as it is in an established city of the Old World or in Johannesburg or Cape Town, but they must reflect with some accuracy what is happening in the developing areas of our own country.

Another example of limitation in our figures is in neurotic and psychological disturbances. At one time specialist care for these conditions was given as part of the service, without charge to the patient. The introduction of a rule by which the member became responsible for 50% of the psychiatrist's fees immediately reduced to one-tenth the number of people requesting this form of treatment. No attempt is made to guess which incidence of psychological disturbance is the correct one—that before or that after the introduction of personal payment.

Again our figures have been affected by the Provincial hospital system of record keeping and destruction, as a result of which a retrospective review of hospital cases has not been a practical or rewarding possibility.

On the other hand, in my opinion the figures here presented are of special value for two main reasons. In the first place, they are based on the records of a homogeneous consulting medical organization serving the greater part of an industrial town. Such records are usually difficult to compile because of the many distinct and unrelated forms of medical care which ordinarily serve a community. It is impossible to gather together and publish in one review the records of separate firms of private doctors, medical and benefit societies, full-time hospital personnel, district surgeons, public-health officials, and local and visiting specialists.

Secondly, the physicians in this service have unusual statistical control for the following reasons:

(*a*) Patients never come to us direct, but are referred through the full-time general practitioners and the other specialists in the service. The services of physicians are provided without restriction to the patient. He may, through us, request not our opinion but that of any private physician of his choice. We have the advantage, therefore, that there is no obstruction, financial or ethical, to any physician's opinion being obtained whenever a patient or his doctor may require it.

(*b*) It is an essential part of our set-up that the reference, the report, and the return, is made to the general practitioner. Continuity of treatment and follow-up should consequently be complete.

(*c*) Because of our responsibility in the service, we full-time physicians cannot allow ourselves to build a reputation in one particular branch of medicine, as a physician in private practice must do.

(*d*) The diagnosis of the physician is for ever afterwards subject to the critical review of the whole team. No one of them, either in day-to-day practice, or in this review of 3,000 cases, has ever spared our feelings.

I have found it a stimulating exercise, and one which contains many surprises, to review what the community really requires of me as a physician—what problems are most likely to require my opinion, what diseases from the patient's and general practitioner's point of view have the greatest frequency of importance, on what subjects one can most usefully spend one's reading, and what groups of cases—fascinating as they may be—are too rare in a normal healthy community to deserve much of one's time. From our younger colleagues' point of view, these considerations throw light on the question of what particular specialization is worth entering into: how many specialists the cases likely to arise in a particular speciality will keep busy in a town, in a province, or in the whole country?

CLASSIFICATION

I have divided my cases according to their major pathology into the system under which the pathology and its presenting symptom have been found to fall. Each case is counted once only. This classification naturally presents difficulties. For instance, a great number of patients present with the complaint of 'chest pain'—straightforward to them but very complex to us. Headache seldom has a single aetiology. Hypertension has been put under the 'heart disease' group, and obesity, so irritatingly frequent a complaint, under the endocrine system. Periodic sickness in children, and enuresis, are classified under psychological disturbances.

The first gross break-down of the 3,132 cases is presented in Fig. 1. It is evident that heart disease, alimentary disease, and disease of the central nervous system (excluding psychological states) provide half the cases seen. It is also evident that anyone setting up practice in blood and peripheral vascular disease alone, which provide 4% of the total of our cases, should find a really big city to settle in! (I must remind the reader that, as this is a review of cases as they come to a physician, it would be wrong to make the claim that these percentages exactly reflect the proportion in which they occur in the population. Many cases—one can instance peptic ulcers—will be cared for by their general practitioners or go direct to the surgeon.) There are many suppressed factors which influence the partition of cases. Heart disease is high not only because the incidence of cases is high but because our general practitioners feel that the decisions to be taken are of such importance that they require the backing of the specialist's opinion; alimentary

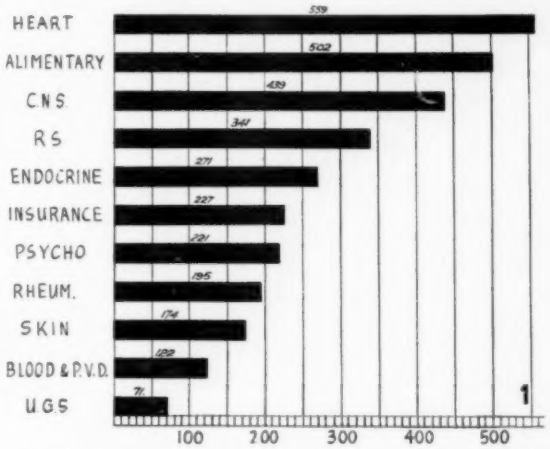

Fig. 1. Break-down of 3,132 consulting-room cases seen by the general physicians. CNS = central nervous system. RS = respiratory system. Psycho = psychological. Rheum. = rheumatic fever. PVD = peripheral vascular disease. UGS = urogenital system.

cases notoriously become impatient of their symptoms; and to the difficulty of diagnosis in central-nervous-system cases is added the importance of their ability (especially in epileptics) to continue work.

From the point of view of follow-up, it is one of the trials of consultant practice that we so seldom know what has happened to our patient once he has left us—has he died, has he thought so little of us that he has taken up faith-healing or, worse still, has he gone to the brilliant young specialist from Cape Town? I can offer some comfort here. He very seldom does any of these things. The follow-up figures available to me show that a large proportion of the cases I see only once (and this group is 30% of my practice) are satisfied with the reassurance the examination brings them, and thereafter do not trouble their practitioner, but get well with time. This reassurance appears to be the physician's greatest usefulness to a community! Only a very small number—less than 1% in the 2,000 or so we have followed-up—develop some serious illness that was not apparent at the time or was missed at examination. An even smaller percentage die without one's knowledge. The dreadful things we envisage in our depressed moments do not therefore appear to happen to our patients.

Anyone who does insurance examinations, and has not the opportunity of follow-up, worries whether his assessment of his patient was correct. It is of interest that for the last few years we have been examining all the 'executives' and new employees of the industry who are over 50 years old. Although we cannot set an insurance-policy standard of fitness where workers are scarce, only 1 of the 208 we have accepted has so far let us down by dying!

HEART DISEASE

The presenting pathology in the 559 cases of heart disease is shown in Fig. 2.

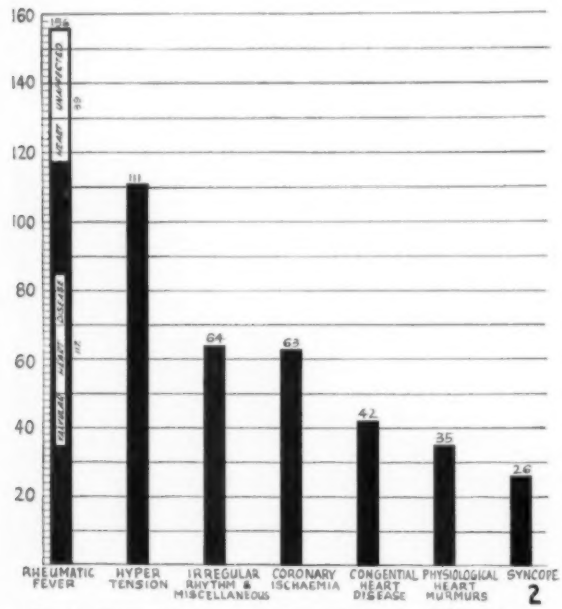

Fig. 2. Heart disease: presenting pathology in 559 cases.

Rheumatic Heart Disease

To me the most disturbing fact that arises is the number of cases of rheumatic heart disease we have. It may be compared with the expected average of 20% of all heart disease as quoted by Paul Wood for Britain—and there have been those who have taught that rheumatic fever is infrequent in South Africa!

It is alarming to think of so great a number of cardiac cripples moving about in a small community like ours. If we have such a number, what will the figure be in Johannesburg or Cape Town, where not every rheumatic child has free access to a physician?

We have compared the number of rheumatic heart cases which have come to us against the certified number of poliomyelitis cases in our community. The figures are: rheumatic fever 155, poliomyelitis 22. Fig. 3 shows the relative effects of these diseases. When one considers the emotions aroused by the child obviously limping in our streets, one wishes that that same emotion would flow for the children lying at home crippled by rheumatic fever, not merely with a handicapped future ahead of them but with each day an increasing battle for mobility and life. Perhaps then we should get our rheumatism foundation.

A second point of great interest is the pool of potential cardiac surgical cases we carry, and how few of them come

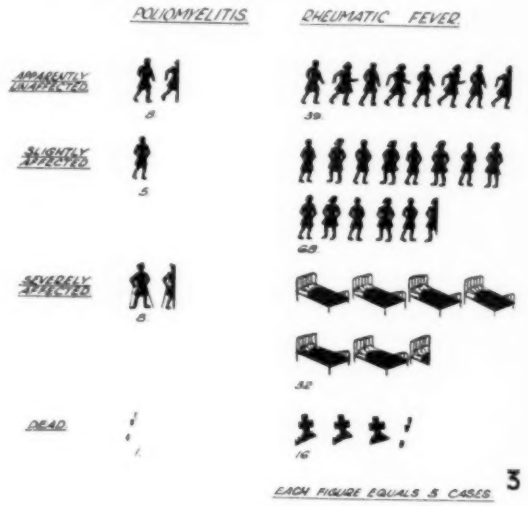

Fig. 3. Incidence and effects of poliomyelitis and rheumatic fever.

to operation. Out of the 117 cases of rheumatic valvular disease only 9 have been operated upon; and out of the 42 cases of congenital heart disease only 3. Some years ago I was asked by our committees to estimate the number of valvular and congenital deformities which might come to operation, so that the necessary financial preparation could be made. I gave my estimate and £5,000 was placed in a fixed deposit. Ever since then the committees have asked me, 'Where are all those mitral stenotics and children with congenital hearts which you said you had?' Well, here they are. Obviously when I prepared my original figure I was looking at the problem from the point of view of a

cardiac clinic. I had failed to recognize the factors which apply when the patient is taken home.

Perhaps this is the greatest lesson I have learnt from my review. The major medical illnesses take on an entirely new slant when one relates them to the life of a community. No longer does one then follow the natural history of a disease, but the history of a community relative to disease. A girl of 16 may be found to be suffering from a patent ductus arteriosus. She is an ideal case for surgery. But she feels she has already got through 16 years pretty well and, anyway, she hopes to marry in 6 months' time and is not going to lose her chance of *that* for any operation. And once she has married and has the support of an unintelligent husband, one may argue for years. This has happened to us with 3 out of 5 cases of patent ductus. Or one may have followed a child with a septal defect or aortic stenosis through the years, waiting for the full development of the heart-lung machine. With the passage of years the child has grown into an age when he must now contribute to the family income and he can no longer be 'laid-off' or 'risked' as he could as a child. Boy-friends and economics destroy statistics. Paul Wood's estimate that 4/5ths of all cases of mitral stenosis will eventually come to surgery is possibly correct, but one must work from the hospital heart clinic, and remember the 'eventually'!

We have reviewed our own cases of heart disease operated upon and find they divide up as follows:

Those operated upon	12
Those unsuitable for surgery at the present time for clinical or technical reasons	41
Those with established mitral or congenital valvular disease but who feel too well to consider surgery	67
Those suitable for surgery, but delaying for one reason or another	9
Those who have moved to another town	30

With surgery freely offered, only 12 out of 159 cases have come to operation!

We frequently refer patients from this group to thoracic surgeons with the request that if they cannot persuade the patient to accept operation, they should establish their own base-line of observation for the future. The patient always seems to take the 'base-line' choice. I should be very interested to hear how far physicians—not thoracic surgeons—feel we should force the surgical issue in these cases.

Coronary Thrombosis

We have had 50 cases of coronary thrombosis, with 6 deaths. Each case has been counted once only; several, dead or still alive, have had more than one thrombosis. We had one young man of 28 with 4 separate thromboses identifiable on one ECG, and more than 20 scars found at post-mortem. All our cases are treated in hospital and all with anticoagulants. Because of their difficulties with laboratory services the private practitioners of the town, who are responsible for about one-quarter of the European population and the entire non-European, do not treat with anticoagulants. From their comparative figures—which, however, are small—it appears that their survival rate for the first month is not significantly worse than ours. The majority of deaths (12) among our own and the private cases have taken place before the patient could be admitted to hospital. We shall follow the comparison as long as the differences in treatment remain. The important point to us, however, is that whether treatment with anticoagulants has been used or not, the mortality rate of the group as a whole is much lower than it was 20 years ago. This, of course, has been noted by many authorities.

ENDOCRINE DISEASES

These are classified in Fig. 4.

We propose making the follow-up of *thyroid disease* the subject of a fuller report later but so far, in consultation with the general practitioners and our patient's families, we are impressed by the number of patients who persist with symptoms of nervousness and tension even after apparently adequate treatment by any of the accepted methods, including thyroidectomy. Indeed, patients made myxoedematous by treatment are often nearly as tense and difficult with their families after therapy as before it. It would seem that to our difficulties of diagnosis in thyroid we should at our first consultation add a psychologist's assessment of the patient's basic personality, for that personality will persist beyond therapy, and our expectation (and promise) of changing a nervous person to a calm one may often prove wrong.

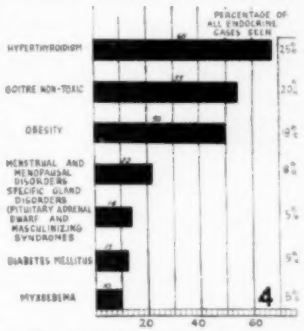

Fig. 4. Endocrine diseases.

DISEASES OF THE CENTRAL NERVOUS SYSTEM

These are classified in Fig. 5.

Epilepsy has been particularly interesting to us because by law no established epileptic is allowed to work in heavy industry. In consequence, to allow our patients every chance before condemning them to medical boarding, we have investigated them fully, usually up to air studies and angiograms.

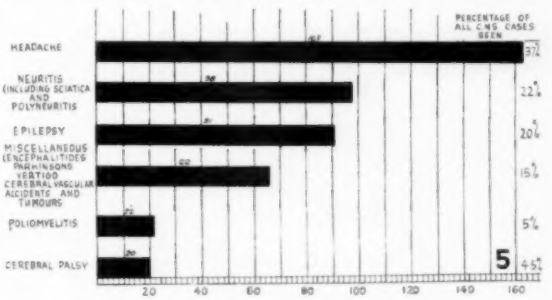

Fig. 5. Diseases of the central nervous system.

It is common practice in a small town to accept a suspect epileptic as such, and for ever afterward he is treated by the general practitioner with sedatives and anticonvulsants. The cases which are referred to neurologists are those which are alarmingly out of true—probably tumours. Because of our interest in their social future, our patients fall between these two groups, the epileptics of the general practitioners and those of the neurologists. This probably explains the

considerable difference in the frequency with which tumours are found among our patients and the figures suggested by some of our neurological colleagues. Although the few text-book estimates place the frequency of brain tumour in epileptics above 2% one occasionally hears it stated that surgery for neoplasm may be expected in anything up to 20% of epileptic presentations. Our experience supports the text-books and not our enthusiastic associates. In our 91 cases investigated and followed-up we have found only 2 tumours, and one of these a secondary from a bronchial carcinoma. Cerebral atrophy as judged by air studies is the most frequent pathology found.

RARE DISEASES

Earlier on I mentioned those fascinating diseases which seemed to occur so infrequently in a community and yet take up so much of our time in hospital practice. In spite of diligent searching and follow-up we have had only one case of lupus erythematosus disseminatus in 8 years, and there have been only 2 cases of collagen disease (indefinitely diagnosed by biopsy), 11 of rheumatoid arthritis (ours is admittedly a population aged largely under 60), 2 of nephrosis, 2 established cases of electrolyte-retaining nephritis, and 8 of subacute bacterial endocarditis (in spite of the large number of rheumatic and congenital hearts). There has been only 1 established case of ulcerative colitis and none of pernicious anaemia. Rare diseases we have had, but here we are discussing the more fashionable diseases.

On the other side of the picture, one wonders where one could best spend one's reading to give the greatest service to the patient group as a whole. And here our figures and our experience are definite. Most of all one needs a clear knowledge and a differential diagnosis beyond the usual on headache, chest pain, colitis in its various forms, thyroid disease and epilepsy—all too common to be exciting, except that we know so desperately little about them.

WORLD MEDICAL ASSOCIATION

PARACELSUS MEDAL AWARDED

Three doctors were awarded the Paracelsus Medal at the joint opening session of the 14th General Assembly of the World Medical Association and the 63rd Deutsche Arztetag which convened in the Berlin Congress Hall, West Berlin, Germany, on 16 September.

The Paracelsus Medal, named for the 16th century physician and chemist who promoted the use of specific remedies and was the author of many medical works, was established in 1952 as the highest award bestowed by the 'Deutsche Ärztetag' (German Physicians' Day). It is usually awarded to 3 distinguished doctors annually at an impressive ceremony.

Those honoured were:

Dr. Curt Emmerich of Baden-Baden, Germany, in recognition of his untiring efforts to picture for the people of the world the true character of the doctors who make up the medical profession. Dr. Emmerich, whose writings are published under the pseudonym of Peter Bamm, is being cited for having established a memorial to the reputation of the medical profession in his book *The Invisible Flag* which has been translated for publication in 8 languages. In this book Dr. Emmerich vividly demonstrates that under the invisible flag of humanity in accordance with the Hippocratic oath, the medical profession, in modern civilization as in ancient times, is at work all over the world at all times protecting life and giving aid to suffering mankind.

Dr. Walter Stoeckel of Berlin, Germany, in recognition of his outstanding contributions to medical science. Dr. Stoeckel, Professor of Obstetrics and Gynaecology, Professor of Urology, and Emeritus Director of the University Women's Clinic, is a wise counsellor, respected teacher and esteemed educator of generations of medical students and doctors. His personality, scientific accomplishments and outstanding descriptive ability in words and writing and surgical dexterity contributed to the cure of thousands of patients. Even during the frustrating trials resulting from the destruction of his clinic during the distress of war and its aftermath, which overtook him in his advancing years, Dr. Stoeckel faithfully devoted his life to the principles of duty of the medical profession and made the welfare of his patients his first and primary consideration.

Dr. Louis H. Bauer of New York City, Secretary General of the World Medical Association, in recognition of the service he has rendered to the medical profession of the world. The citation notes Dr. Bauer's outstanding service in promoting freedom and upholding the honour of the medical profession in scientific, social and economic aspects. However, the Paracelsus Medal was awarded to him in recognition of the valuable contribution that he has made in the administration of the global organization of the doctors of the world and his friendship toward the German physicians.

VRAE BEANTWOORD : QUESTIONS ANSWERED

COLLAPSE AFTER DRUG THERAPY

Q—*For some time I have been using a combination of 'pethidine' 50 mg. and 'largactil' 50 mg. by intramuscular injection in cases of severe headache associated with nausea and vomiting, where no true diagnosis has been made. I have had really excellent results.*

However, another general practitioner has asked me if I have not yet had any patient collapse on this treatment. He had a patient who collapsed, with an impalpable pulse and an unrecordable blood pressure. (Fortunately the patient recovered.)

Do you think that this collapse was due to the fact that the two drugs were used in combination, or to a hypersensitivity on the part of the patient to either the 'largactil' or the 'pethidine'?

A—'Pethidine' may cause dizziness (in 60% of patients), nausea, vomiting and other features. Intramuscular injection of 100 mg. has caused cyanosis and an unrecordable blood pressure. Injection into a vein produces a fall in blood pressure; an asthmatic attack has been precipitated in this way. Nalorphine or levallorphan may be used as antidotes. 'Pethilorfan' contains levallorphan.

'Largactil', 25 - 50 mg., by intramuscular injection may produce toxic effects including transient faintness, flushing of the face, palpitations, dizziness, lightheadedness, tachycardia and moderate hypotension. Faintness may last for some hours after each dose of the drug. Acute hypotension after large doses may be severe or even fatal. As an antidote to severe hypotension, noradrenaline may be given.

Since both the drugs mentioned may be the cause of a collapse, either may have been to blame in the case mentioned. There is no evidence in the literature of a synergistic effect if both drugs are used together.

Incidentally, 'benadryl', 'dramamine' and 'marzine' appear to be more useful than 'largactil' in combating nausea and vomiting caused by drugs.

Readers are invited to submit questions for this column to the Editor, *South African Medical Journal*, P.O. Box 643, Cape Town.

SOUTHERN AFRICAN CARDIAC SOCIETY: CONGRESS

The Southern African Cardiac Society will hold a Congress in Cape Town on 19 - 22 October 1960. A preliminary programme is reproduced below for the information of readers of the *Journal:*

PRELIMINARY PROGRAMME

Wednesday 19 October, Medical School, University of Cape Town

2 p.m. Welcoming address by Dr. Maurice Nellen, National President of the Society
Registration

Electrocardiography and Phonocardiography

The effort electrocardiogram: Drs. M. M. Zion and B. A. Bradlow
Paroxysmal tachycardia with special reference to repetitive paroxysmal tachycardia: Dr. A. D. Charters
Aberrant ventricular conduction: Dr. L. Schamroth
The electrocardiogram of the newborn — an inter-racial study: Drs. V. Schrire, G. Sutin and J. Rabkin
Discussion
Auscultatory findings in systemic hypertension: Dr. J. B. Barlow
Mid-systolic clicks: Dr. J. V. O. Reid
Heart murmurs in the frog: Dr. K. Furman
Discussion

General

Pulmonary function and thyrotoxicosis: Dr. H. P. Wassermann
Cardiac tone: Dr. W. Davis
Discussion
Fluid-volume control in open-heart surgery: Dr. W. F. Scott
Some observations on trichlormethiazide: Drs. N. Levin and B. Goldberg
Repoft on cases treated with Ismelin: Dr. B. Koch
Discussion

Thursday 20 October, Medical School, University of Cape Town

Symposium on the Effects of Vaso-active Drugs on Cardiac Haemodynamics and Murmurs

From the Cardiac Clinic, Groote Schuur Hospital, Cape Town, and the CSIR Cardiopulmonary Unit, University of Cape Town Medical School. Papers to be presented by Drs. V. Schrire, M. Nellen, L. Vogelpoel, A. Swanepoel and W. Beck.

8.45 a.m. Introduction
The effects of amyl nitrite on the cardiac haemodynamics in normal man
The effects of amyl nitrite on the murmurs of pulmonary stenosis, tetralogy, and small ventricular septal defects
The effects of amyl nitrite on the magnitude of the flows and shunts in pulmonary stenosis, tetralogy, and small ventricular septal defects
The effects of phenylephrine on the cardiac haemodynamics in normal man
The effects of phenylephrine on the murmurs of pulmonary stenosis, tetralogy, and small ventricular septal defects
The effects of phenylephrine on the flows and magnitude of shunts in pulmonary stenosis, tetralogy and small ventricular septal defects
Intracardiac phonocardiographic recording of the effects of amyl nitrite and phenylephrine on the murmurs of pulmonary stenosis, tetralogy, and small ventricular septal defects
Discussion

10.30 - 10.45 a.m. Tea

Symposium on Techniques of Cardiac Investigation

A simple method of arterial puncture: Drs. B. Kaplan and V. Schrire
The use of dye-dilution curves in the diagnosis of congenital heart disease: Drs. W. Beck, V. Schrire, M. Nellen, L. Vogelpoel, and A. Swanepoel
Ciné-angiography of the heart and great vessels: Drs. M. B. M. Denny and P. Marchand
Retrograde aortic catheterization: Drs. A. Swanepoel, W. Beck, M. Nellen, L. Vogelpoel and V. Schrire
Discussion

12.50 - 2.00 p.m. Luncheon

Symposium on Congenital Heart Disease

Atrial septal defect: Mr. D. Fuller *et al.*
Closure of atrial septal defect under hypothermia: Dr. A. J. P. Graham
Discussion
Endocardial cushion defects: Drs. C. N. Barnard and V. Schrire
Discussion

3.50 - 4.10 p.m. Tea

Patent ductus arteriosus in infancy with special reference to pulmonary hypertension: Drs. L. Braudo, S. Javett and M. M. Zion
Corrected transposition of the great vessels: Drs. W. Beck, V. Schrire, M. Nellen, L. Vogelpoel and A. Swanepoel
Organic functional pulmonary atresia: Drs. V. Schrire, G. Sutin and C. N. Barnard
Discussion
Cyanotic spells in Fallot's tetralogy: Drs. L. Braudo and M. M. Zion
Relative pulmonary stenosis: Drs. M. Nellen, V. Schrire, L. Vogelpoel, A. Swanepoel and W. Beck
Pulmonary incompetence: Dr. L. H. Klugman
Discussion

Friday 21 October, Medical School, University of Cape Town

Myocardial Disease

8.45 a.m. The heart in kwashiorkor: Drs. P. M. Smythe, A. Swanepoel and J. Campbell
Familial heart disease: Dr. J. M. Combrink
Discussion
Types of cardiac disease in the elderly African: Dr. M. Gelfand
The pathogenesis of hyaline arteriosclerosis: Dr. B. McKinney
Acute rheumatic fever: Drs. H. Utiane and L. Braudo
Discussion

10.30 - 10.45 a.m. Tea

Valve Disease

Posterior mitral cusp incompetence: Dr. J. C. van der Spuy
Silent mitral incompetence: Drs. V. Schrire, L. Vogelpoel, M. Nellen, A Swanepoel and W. Beck
Discussion
The diagnosis of atrial myxoma: Dr. J. B. Barlow
Surgery of aortic stenosis: Mr. W. Phillips
Discussion
Experimental observation of profound hypothermia for open-heart surgery: Mr. P. Marchand
Clinical observations on profound hypothermia for open-heart surgery: Drs. C. N. Barnard and J. Terblanche

12.50 - 2.00 p.m. Luncheon

Nutritional Research Unit, Groote Schuur Hospital

2.00 p.m. The relationship of smoking to ischaemic heart disease
1. Possible mechanisms: Dr. B. Bronte-Stewart
2. Taste thresholds: Dr. L. H. Krut
3. Food preferences: Miss M. J. Perrin

Blood-group pattern in ischaemic heart disease: Dr. M. C. Botha
Plasma fatty acids and atherosclerosis: Dr. G. Young
Depot fat composition in the three racial groups and coronary subjects: Dr. L. H. Krut
Fat-tolerance studies in ischaemic heart disease: Dr. I. A. D. Bouchier
Active principles in egg-yolk lipid: Messrs. V. Wells and J. A. Wilkens

Saturday 22 October, Karl Bremer Hospital, Bellville

9.30 a.m. Main lecture hall, Clinical Buildings
The serum-protein spectrum in 100 normal Bantu males:

Dr. P. D. R. van Heerden
Cardiological applications of the artificial kidney: Dr. P. W. Botha
Studies on oxygen consumption of respiratory muscles and oxygen utilization in tissue: Dr. H. P. Wassermann
The artificial heart: Dr. B. J. v. R. Dreyer (film)
Exercise-tolerance tests: Dr. L. Potgieter
Tea
Scientific Exhibitions and Informed Discussions
Cardiac pathology: Dr. H. W. Weber
Some improvements and modifications to the Kay Cross heart-lung oxygenator: Dr. B. J. v. R. Dreyer
Some electrocardiograms of special interest
Display of the results of some of the work discussed in the papers presented.

APPOINTMENT OF SECRETARY FOR HEALTH

On 3 October the Minister of Health, Dr. J. A. M. Hertzog, announced the appointment of Dr. Bernard Maule Clark, Deputy Chief Medical Officer of the Department of Health, as Secretary for Health and Chief Health Officer for the Union. The appointment is with effect from 3 August, when Dr. Clark's predecessor, Dr. J. J. du Pré le Roux, retired.

Dr. Clark joined the Department of Health in 1936 as Assistant Health Officer attached to the Cape Town office. At that time the Chief Health Officer and Secretary for Health was Sir Edward Thornton. Before joining the Department Dr. Clark had been Medical Officer of Health at Pietermaritzburg and had also been in private practice in Pretoria.

After serving in Cape Town for some years, Dr. Clark was transferred to Johannesburg as Senior Assistant Health Officer in the Department of Health and subsequently moved to Pretoria as Deputy Chief Health Officer. He has been Deputy to Dr. du Pré le Roux since 1952 and has been Acting Secretary for Health since Dr. le Roux's retirement.

IN DIE VERBYGAAN : PASSING EVENTS

Dr. I. P. Jaffe, of Cape Town, has recently returned from a visit overseas where he attended the Great Ormond Street Hospital for Sick Children, London, and the Children's Hospital in Zurich, Switzerland. Dr. Jaffe has now resumed practice at 506 Medical Centre, Heerengracht, Cape Town. Telephone 2-8506.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 24 October at 5.10 p.m. in the Institute Lecture Theatre. The film 'Lymphomas and leukaemias' will be shown by courtesy of the National Cancer Association of South Africa.

* * *

Symposium on Parenteral Fluid Therapy. A symposium on parenteral fluid therapy has been arranged by *Medical Proceedings*. This will be held in Johannesburg on 28 and 29 October 1960 in the Lecture Theatre, 2nd floor, South African Blood Transfusion Service, cor. Klein and Esselen Streets, Hillbrow, Johannesburg.

The symposium will include sessions on surgery, neurosurgery, paediatrics and artificial dialysis. The hours of the symposium are from 10 a.m. to 5 p.m.

Interested medical practitioners are invited to attend. They should intimate their decision to attend the symposium as early as possible to: Dr. H. A. Shapiro, Editor, *Medical Proceedings*, P.O. Box 1010, Johannesburg.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 19 October at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Drs. C. N. Barnard and J. Terblanche will speak on 'Hypothermia'. All members of the Medical Association are welcome to attend this lecture.

* * *

Dr. and Mrs. J. J. Jacobson, of Cape Town, have left on a vacation and study trip to England, the Continent, and Israel and will return to the Union at the end of November.

* * *

Vaal River Branch (M.A.S.A.). Dr. C. G. S. van Heyningen, of Vereeniging, has been elected to represent this Branch on the Federal Council of the Association.

* * *

Dr. C. H. H. Coetzee ('Oom Harry'), of Pretoria, has requested that his name be removed from the Specialist Register and will, in future, confine himself to *locum tenens* appointments and anaesthetics, as a general practitioner.

* * *

Dr. R. Lang, has returned to Cape Town after a visit overseas where he attended various universities and hospitals in the USA, Canada, and London.

* * *

College of General Practitioners, Cape of Good Hope Faculty. This Faculty is holding a Refresher Course at the Conradie Hospital, Pinelands, Cape, with the kind permission of the Superintendent, on 22 and 23 October 1960. Doctors wishing to attend the course should communicate with the Secretary, Cape of Good Hope Faculty of the College of General Practitioners, P.O. Box 643, Cape Town, or telephone 3-2022. A fee of £1 1s. for the course, or part thereof, will be charged to non-members.

FARMASEUTIESE NUUS : PHARMACEUTICAL NEWS

DIFFERENTIAL DIAGNOSIS FOR PRACTITIONERS IN THE TROPICS : A CLINICAL HANDBOOK

FBA Pharmaceuticals (S.A.) (Pty.) Ltd. are distributing the book *Differential Diagnosis for Practitioners in the Tropics: A Clinical Handbook* by W. H. Jopling, M.R.C.P., D.T.M. & H., in collaboration with Philip Manson-Bahr, free of charge to practitioners in South Africa. Doctors wishing to obtain a copy of this book, which is not for sale, are asked to write to Messrs. FBA Pharmaceuticals (S.A.) (Pty.) Ltd., P.O. Box 10233, Johannesburg.

This book comprises 159 pages and is well illustrated. It is published by the Scientific Department, Pharmaceutical Division of Farbenfabriken Bayer AG of Leverkusen, Germany.

'The main object of this publication is to act as a guide for the general practitioner in the tropics' where ancillary diagnostic services are not available. Particular attention is paid to the prevalence of parasitic infections which may complicate a diagnosis and emphasis is placed on the clinical examination of the patient, by inspection and palpation, rather than by the use of intricate diagnostic procedures.

Readers wishing to obtain more information about this book before requesting a copy from the distributors, are advised to consult A. R. D. Adams' excellent review which was published in *Tropical Diseases Bulletin*, vol. 57, p. 863, of August 1960.

NEW MEDICAL FILM

SKF Laboratories have available on loan, without charge, a film entitled 'Resuscitation of the newborn', which is intended for showing to professional audiences only. It is a 16 mm. sound film in colour and the running time is 25 minutes.

This film illustrates the essential principles involved in the resuscitation of infants who do not breathe — or whose respira-

tion is impaired — at birth. The procedures shown are those developed by the Special Committee on Infant Mortality of the Medical Society of the County of New York. Through live-action delivery-room photography of actual resuscitations and through animation, the procedures and apparatus necessary for effective resuscitation are shown. The Apgar scoring system by which the clinical status of the infant can be rapidly assessed is demonstrated. The physiology of pre- and postnatal oxygenation and the factors that may contribute to producing respiratory depression in the newborn are described.

Bookings for this film may be arranged through local SKF representatives or by writing to SKF Laboratories, P.O. Box 38, Isando, Transvaal. If possible, 4 weeks' prior notice and an alternative showing date of at least 1 month after the preferred date should be given.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

MEXAFORM

Ciba Limited, of Basle, Switzerland, introduce Mexaform, a new antidiarrhoeal agent, and supply the following information:

Mexaform is a potent intestinal antiseptic containing Vioform and Entobex, two antibacterial and antiparasitic substances whose effects supplement and reinforce one another in ideal fashion. Vioform, a drug long acknowledged as an eminently dependable intestinal antiseptic, displays a broad range of antibacterial and antiparasitic activity. Entobex exerts a specific effect on pathogenic protozoa of various types (amoeba, giardia, trichomonas, chilomastix) and also possesses good antibacterial properties. Mexaform contains, in addition, Antrenyl, a spasmolytic, which serves to combat the spasm, colic, and tenesmus frequently occurring in diseases associated with diarrhoea. Mexaform is excellently tolerated and can also be prescribed for children.

Indications. Mexaform is indicated for all types of diarrhoea, including especially diarrhoea of infectious origin due to bacteria or protozoa, as well as unspecific diarrhoea or diarrhoea of uncertain aetiology and summer diarrhoea. Mexaform may be employed both in the acute stage of the disease and in chronic or relapsing cases.

Administration and dosage. The average dosage for adults is 1 tablet of Mexaform 3 times daily; in severe cases, treatment can be initiated with 2 tablets 3 times daily. Children should be given 1 tablet of Mexaform 1-3 times daily, depending on the child's age. A few days' treatment is generally sufficient. In areas in which amoebiasis is common, it is advisable to continue the treatment for 1 week, even where pathogens can no longer be traced in the stools and where a symptomatic improvement or a clinical recovery has already been achieved.

Mexaform is particularly suitable as prophylactic treatment against diarrhoeal infections, e.g. when travelling to a hot climate or when otherwise exposed to the risk of intestinal infection. For this purpose, it is sufficient to take 1 tablet 2 or 3 times daily.

Each tablet of Mexaform contains 200 mg. iodochlorhydroxyquinoline + 20 mg. 4,7-phenanthroline-5,6-quinone + 2 mg. methyl bromide of the diethylamino-ethyl ester of phenylcyclohexyl-hydroxyacetic acid.

Packing. Mexaform is supplied in packs of 20 and 100 tablets.

Further information may be obtained from Ciba (Pty.) Ltd., P.O. Box 5383, Johannesburg. See also advertisement on p. xv.

HIRUDOID

Newport Trading Corporation (Pty.) Ltd., announce the introduction of Hirudoid, manufactured by Luitpold Werke, of Munich, Germany, and supply the following information:

Therapy with Hirudoid aims at inhibiting and shortening the rigor, calor and dolor of locally circumscribed inflammatory processes, which frequently accompany thrombotic and phlebitic conditions, but above all aims at the dissolution of blood clots.

Hirudoid is a standardized organic preparation with heparinoids as active principles contained in a white neutral ointment base. Extensive clinical experience as well as experimental laboratory tests with Hirudoid prove that a percutaneous absorption of the heparinoids takes place, and it is of particular importance to stress that the organic substances of the product do not require the presence of a skin-irritating agent to permeate. The standards against which the anticoagulant effect of Hirudoid is experimentally measured are the known coagulation times of human blood. An average (objective) prolongation of the clotting time by 52% was found to be the case as a result of topical application of Hirudoid, when an optimal specific effect was seen about 6 hours thereafter. Whilst an undesirable (sometimes unpleasant) pronounced hyperaemizing effect is avoided, the full absorption of the specifically active substances brings about the rapid remission of subjective symptoms (pressure, pain). In addition to the anticoagulant effect, a noticeable antiphlogistic and fibrinolytic action starts with the first application.

Hirudoid is indicated for topical use in traumatic and pathological conditions near the surface of the skin such as: haematomas, contusions, thrombosis, phlebitis, thrombophlebitis, varicose veins, ulcus cruris, furuncles, and inflammatory infiltrations due to injections.

Hirudoid is applied in a layer or on a mousselin pad on the inflamed or thrombotic surface or around the open ulcer itself once or twice a day, until the condition has subsided. It has no side-effects.

Hirudoid is supplied in tubes of ½ oz., 1½ oz., and 5½ oz.

Further information and samples may be obtained from the importers: Newport Trading Corporation (Pty.) Ltd., P.O. Box 1871, Johannesburg. See also advertisement on p. xxii.

BOEKBESPREKINGS : BOOK REVIEWS

PSYCHIATRY IN GENERAL PRACTICE

Psychiatry in General Practice. By J. A. Weijel, M.D. Pp: viii + 208. 37s. 6d. Amsterdam, London, New York, Princeton: Elsevier Publishing Company. 1958.

The author has worked in a research team with the famous Dr. Querido of Amsterdam, who demonstrated conclusively that many of the patients admitted to general hospitals had psychological and social problems about which they did not complain. In this book the way in which a patient's medical problem is interwoven with psychiatric problems is explored. As psychiatric knowledge becomes better used in general hospitals, inevitably general practitioners themselves will want to obtain understanding about mental mechanisms and will want to train themselves in psychiatric treatment techniques. The book deals with the nature of the general practitioner's approach to the patient and the patient's own attitude to medical treatment, and it attempts to make psychiatric methods available for general practitioners.

In this difficult task the author is fairly successful. He holds that while a vast amount of knowledge about the psyche is already available, it is not put into a form the non-specialist is able to use. The general practitioner cannot depend on classical descriptions of the transference situation, for the emotional attachment of a mentally normal patient towards his doctor is very different from the transference of a frankly psychiatric patient. The general practitioner must examine his patient physically and, the author stresses, need not deter himself with the consideration a psychotherapist would entertain (that he might be acting seductively towards the patient). It is not helpful for the general practitioner to reflect when he injects his patient that he might be acceding to coital phantasies in the patient's mind. General practice is totally different from an out-patient psychiatric department.

However, when doctors become more widely aware that neurotic complaints can be hidden in physical disorders, and perceive the origins of neurosis in the problems of life itself—the stresses

of living with others, of civilization and culture—the conventional medical response of reducing a 'case' to a somatic level is no longer possible. The doctor then needs the techniques of opening the 'case' to include the psychosocial aspects of the patient's existence. The author has attempted to provide interested doctors with a psychosocial 'stethoscope' for discovering the psychological aspects of the patient's life. Few doctors reading his book will remain unimpressed with the inadequacy of traditional medical approaches in general practice. H.W.

PLASTIC PROCEDURES IN GENERAL SURGERY

Fundamental Technique of Plastic Surgery and their Surgical Applications. By I. A. McGregor, M.B., F.R.C.S. (Eng.), F.R.F.P.S. (Glas.). Pp. viii + 244. Illustrated. 30s. net + 1s. 7d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1960.

This book is not, as its title suggests, intended for the plastic surgeon, but should prove a valuable guide to the general surgeon who is frequently confronted with conditions in which skin coverage is necessary.

The procedures dealt with are clearly described and well illustrated, and they cover a wide field of surgery. The chapter on eyelids should be of considerable interest to the ophthalmic surgeon, while that on injury to the hand will provide food for thought for surgeons engaged in accident work.

The author makes a strong plea for immediate skin coverage of the hand and fingers which have suffered skin loss. Prompt treatment on these lines will minimize infection and establish function in a relatively short period, and avoid the frozen hand so often seen in neglected cases.

That surgical bugbear 'the decubitus ulcer' is also discussed and its treatment well illustrated. The author should be congratulated on covering so wide a field is so clear a manner. N.P.

BRIEWERUBRIEK : CORRESPONDENCE

DOCTOR TO DOCTOR OR THE PROBLEM OF REWARD

To the Editor: The question whether a doctor should pay or otherwise suitably reward a colleague whose professional services he has sought, has long been a somewhat vexed and slightly unreasonable subject.

We are brought up — in a medical sense — to regard ourselves as the highly altruistic members of a selfless and learned profession. With such a slightly Sir Galahad concept of ourselves we set out upon our various medical careers. As time passes — especially if we later enter private practice — we finally discover that for practical purposes we are in 'business'. We also learn that the shining armour of some of our medical knights is decidedly less pristine than we originally thought — and hoped.

Thus it is that cash and fees loom larger and larger in our lives and, since expenses steadily mount (more rapidly than fees), we become more and more conscious of our financial needs and problems.

That the economics of medical practice play a large part in our lives is repeatedly reflected in the Correspondence Columns of our *Journal* and in the deliberations of the various councils of our Association; e.g. how to get the medical insurance companies in line with medical aid societies; how to extract our just dues on third-party claims; these and many other monetary matters continually exercise our thoughts, individually and collectively.

Some feel that the very mention of money is indecent; that all lucre is filthy and that on no account should it be allowed to contaminate us. Others, more realistic, feel that it should be seriously discussed whenever necessary and without shame or fear of being thought either miserly or money-grubbing. Still others, though they do not say so, consider its unremitting pursuit to be practically the be all and end all of life.

But when it comes to dealing with colleagues the vast majority of us throw up our hands in horror if a fee is mentioned for our services. For full-time hospital doctors this is a reasonable attitude, since they cannot in any case charge private fees; they are still being paid for the use of their time and they incur no expense to themselves no matter what procedures may be carried out.

For the self-employed doctor who is consulted privately the situation is very different. Certainly the consultation will cost him time, and time, we know, is money. Nevertheless, part from running the risk of missing paying work, he is not necessarily out of pocket; it has not actually cost him money — so far.

Suppose the sick doctor lives out of town, then the specialist or whoever it is he calls upon may have to travel many miles to his bedside, and petrol, as we know too well, must be paid for.

A great many sick doctors, not unnaturally, look after themselves up to a point, and during this period they quite often call directly upon their ancillary specialist colleagues, particularly pathologists and radiologists. Those who have close dealings with doctors in these two specialities know that their overhead and running costs are very high — especially for the radiologists.

Thus it comes about that some of our colleagues have to dip quite deeply into their pockets to help us when we are sick. Not all of us unfortunately seem to realize this, and often a phone call or a short note (or even less) is all the thanks and acknowledgement that we bestow.

What is the solution? Probably the best would be for all of us to belong to a medical aid scheme. At present we do nothing or give presents according to our various tastes, or make a contribution to the Benevolent Fund of the Association.

A colleague has devised a compromise system whereby he sends a cheque to the doctor who treated him for an amount close to that specified in the Tariff of Fees for Approved Medical Aid Societies for the service, accompanied by a letter explaining his 'odd' behaviour. He points out that he is only doing what he would like to have done unto him. He requests a receipt as this can then be used for income tax purposes; whereas receipts from bottle stores — no matter how much whiskey you may give your doctor — are naturally not acceptable to the Receiver of Revenue. This colleague feels that a small cheque is of more use to most of us than another bit of silver to be cleaned.

There can be little doubt that some such system would greatly simplify the dealings of doctor with doctor and make for greater equity.

When Mr. Eric Louw introduced the £100 allowance for medical expenses, that was the moment when our profession should have got its own doctoring house in order. That we did nothing is typical of our dilatoriness and unreal approach to money. However, it's never to late to mend, so let us start now.

Medicus

30 September 1960

MELANOGENESIS : THE MECHANISM OF SKIN PIGMENTATION

To the Editor: I should be glad if you would spare me a little space to correct a statement which appeared in my article on melanogenesis[1] in the issue of the *Journal* for 3 September.

At the top of the second column on page 756 I wrote that mercury and gold 'unite avidly with copper'. I should have written that these two metals can displace copper from tyrosinase thus rendering it inert. The other substances BAL, sulph-hydryl groups, etc. on the other hand probably form bonds with the copper producing the same result.

I apologize for the occurrence of this error which must have offended any biochemical reader.

C. Kevin O'Malley

501 Geneva House
Church Square
Cape Town
27 September 1960

1. O'Malley, C. K. (1960): S. Afr. Med. J., **34**, 753.